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FILE LAST UPDATED: 21 Nov 2000 (20001121/ED)

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=> D STAT QUE L6

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON 521-17-5/RN
L2 12 SEA FILE=REGISTRY ABB=ON PLU=ON ENVM/BI
L3 39 SEA FILE=REGISTRY ABB=ON PLU=ON FABI/BI
L4 13 SEA FILE=REGISTRY ABB=ON PLU=ON (ENOYL-/CN OR "ENOYL-(ACYL CARRIER PROTEIN) REDUCTASE"/CN OR "ENOYL-(ACYL-CARRIER PROTEIN) REDUCTASE (CHLAMYDIA MURIDARUM STRAIN NIGG GENE TC0380)"/CN OR "ENOYL-(ACYL-CARRIER PROTEIN) REDUCTASE (CHLAMYDOPHILA PNEUMONIAE AR39 STRAIN AR39 GENE CP0349)"/CN OR "ENOYL-(ACYL-CARRIER PROTEIN) REDUCTASE (PSEUDOMONAS AERUGINOSA STRAIN PAO1 GENE FABI)"/CN OR "ENOYL-(ACYL-CARRIER-PROT EIN) REDUCTASE (NADH) (BUCHNERA STRAIN APS GENE FABI)"/CN OR "ENOYL-(ACYL-CARRIER-PROTEIN) REDUCTASE (NADH) (AQUIFEX AEOLICUS GENE FABI)"/CN OR "ENOYL-(ACYL-CARRIER-PROTEIN) REDUCTASE (NADH) (CAMPYLOBACTER JEJUNI STRAIN NCTC 11168 GENE FABI)"/CN OR "ENOYL-(ACYL-CARRIER-PROTEIN) REDUCTASE (NEISSERIA MENINGITIDIS STRAIN MD58 GENE NMB0336)"/CN OR "ENOYL-ACP REDUCTASE"/CN OR "ENOYL-ACP REDUCTASE (ENR-A) (ARABIDOPSIS THALIANA GENE AT2G05990)"/CN OR "ENOYL-ACP REDUCTASE (ESCHERICH IA COLI CLONE PEAR3 GENE ENVM)"/CN OR "ENOYL-ACP REDUCTASE (NEISSERIA MENINGITIDIS STRAIN Z2491 GENE FABI)"/CN OR "ENOYL-ACYL CARRIER PROTEIN REDUCTASE (DEINOCOCCUS RADIODURANS STRAIN R1 GENE DR1967)"/CN OR "ENOYL-ACYL-CARRIER PROTEIN REDUCTASE (CHLAMYDIA PNEUMONIAE GENE FABI)"/CN OR "ENOYL-ACYL-C ARRIER PROTEIN REDUCTASE (CHLAMYDIA TRACHOMATIS GENE FABI)"/CN)

L5 1169 SEA FILE=HCAPLUS ABB=ON PLU=ON L1 OR L2 OR L3 OR L4
L6 21 SEA FILE=HCAPLUS ABB=ON PLU=ON L5(L)(ESCH? OR COLI OR SALMONEL? OR TYPHIM?)

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=> D IBIB ABS HITRN L6 1-21

L6 ANSWER 1 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1999:487416 HCAPLUS
DOCUMENT NUMBER: 131:134684
TITLE: Enoyl-ACP (acyl carrier protein) reductase-interacting substances in antimicrobial screening
INVENTOR(S): Levy, Stuart B.; Mcmurry, Laura M.
PATENT ASSIGNEE(S): Trustees of Tufts College, USA
SOURCE: PCT Int. Appl., 80 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9937800	A1	19990729	WO 1999-US1288	19990122
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9923324	A1	19990809	AU 1999-23324	19990122
EP 1049799	A1	20001108	EP 1999-903262	19990122
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-72244 19980123
US 1998-13440 19980126
WO 1999-US1288 19990122

AB Methods and mutants for identifying an antimicrobial compd. which interacts with an ER (enoyl-ACP reductase) polypeptide are disclosed. In particular, the method pertains to screens for identifying an antimicrobial compd. using FabI or InhA mutant cells or polypeptides.

IT **148998-18-9P**, Protein (**Escherichia coli** clone pHAP1 gene envM reduced)
RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); BPR (Biological process); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); PROC (Process)
(amino acid sequence; enoyl-ACP (acyl carrier protein) reductase-interacting substances in antimicrobial screening)

REFERENCE COUNT: 10
REFERENCE(S): (1) Anon; 1996, 7, HCAPLUS
(2) Anon; 1997, 19, HCAPLUS
(4) Bergler, H; EURPEAN JOURNAL OF BIOCHEMISTRY 1996, V242(3), P689 HCAPLUS
(5) Blanchard, J; ANNUAL REVIEWS OF BIOCHEMISTRY 1996, V65, P215 HCAPLUS
(8) Sacchettini, J; US 5702935 A 1997 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1999:398641 HCAPLUS
DOCUMENT NUMBER: 131:180620
TITLE: Cloning and characterization of the gene encoding Pasteurella haemolytica FnrP, a regulator of the Escherichia coli silent hemolysin SheA
AUTHOR(S): Uhlich, Gaylen A.; McNamara, Peter J.; Iandolo, John J.; Mosier, Derek A.

CORPORATE SOURCE: Department of Diagnostic Medicine/Pathobiology, College of Veterinary Medicine, Kansas State University, KS, 66506, USA
SOURCE: J. Bacteriol. (1999), 181(12), 3845-3848
CODEN: JOBAAY; ISSN: 0021-9193
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A Pasteurella haemolytica A1 gene was identified from a recombinant library clone that expressed hemolysis in host Escherichia coli cells. The gene, designated fnrP, had sequence identity to E. coli fnr, a global transcriptional regulator of genes required for conversion to anaerobic growth. FnrP complemented anaerobic deficiencies of a fnr-null mutant strain of E. coli and increased expression of the Fnr-dependent, anaerobic terminal reductase gene,frdA. FnrP was purified, identified by immunoblotting, and shown to be nonhemolytic. When FnrP was expressed in E. coli .DELTA.sheA, a null mutant of the cryptic hemolysin SheA, the transformants were nonhemolytic, indicating that FnrP activates this silent hemolysin.

IT **239448-02-3**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; cloning and characterization of the gene encoding Pasteurella haemolytica FnrP, a regulator of the **Escherichia coli** silent hemolysin SheA)

IT **200385-48-4**, GenBank AF033119
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; cloning and characterization of the gene encoding Pasteurella haemolytica FnrP, a regulator of the **Escherichia coli** silent hemolysin SheA)

REFERENCE COUNT: 30
REFERENCE(S):
(4) Clinkenbeard, K; Am J Vet Res 1991, V52, P453 HCAPLUS
(5) Confer, A; Can J Vet Res 1990, V54, PS48 HCAPLUS
(7) del Castillo, F; Mol Microbiol 1997, V25, P107 HCAPLUS
(8) Dyer, D; Appl Environ Microbiol 1983, V46, P283 HCAPLUS
(9) Fedorova, N; Infect Immun 1997, V65, P2593 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1999:35829 HCAPLUS
DOCUMENT NUMBER: 130:164857
TITLE: The x-ray structure of Escherichia coli enoyl reductase with bound NAD+ at 2.1 .ANG. resolution
AUTHOR(S): Baldock, Clair; Rafferty, John B.; Stuitje, Antoine R.; Slabas, Antoni R.; Rice, David W.
CORPORATE SOURCE: Krebs Institute for Biomolecular Research, Department of Molecular Biology and Biotechnology, The University of Sheffield, Sheffield, S10 2TN, UK
SOURCE: J. Mol. Biol. (1998), 284(5), 1529-1546
CODEN: JMOBAK; ISSN: 0022-2836
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Enoyl-[acyl carrier protein (ACP)] reductase (I) catalyzes the last reductive step of fatty acid biosynthesis, reducing an enoyl-ACP to an acyl-ACP with NAD(P)H as cofactor. The crystal structure of I from E. coli was detd. to 2.1 .ANG. resoln. using a combination of mol. replacement and isomorphous replacement and refined using data from 10 to 2.1 .ANG. to an R-factor of 0.16. The final model consisted of the 4 subunits of the tetramer, wherein each subunit was composed of 247 of the expected 262 residues, and an NAD cofactor for each subunit of the tetramer contained in the asym. unit plus a total of 327 solvent mols.

There were 10 disordered residues per subunit which formed a loop near the nucleotide-binding site which may become ordered upon substrate binding. Each monomer was composed of a 7-stranded parallel .beta.-sheet flanked on each side by 3 .alpha.-helixes with a further helix lying at the C-terminus of the .beta.-sheet. This fold was highly reminiscent of the Rossmann fold, found in many NAD(P)H-dependent enzymes. Anal. of the sequence and structure of I and comparisons with the family of short-chain alc. dehydrogenases, identified a conserved Tyr and Lys residue as important for catalytic activity. Modeling studies suggested that a region of the protein surface that contains a no. of strongly conserved hydrophobic residues and lies adjacent to the nicotinamide ring, forms the binding site for the fatty acid substrate. (c) 1998 Academic Press.

IT **37251-08-4D**, Enoyl-[ACP] reductase, complexes with NAD
RL: PRP (Properties)
(crystal structure of **Escherichia coli** enoyl-[ACP] reductase complexed with NAD)

REFERENCE COUNT: 13
REFERENCE(S):
(4) Banerjee, A; Science 1994, V263, P227 HCAPLUS
(6) Bergler, H; J Biol Chem 1994, V269, P5493 HCAPLUS
(7) Slabas, A; Biochim Biophys Acta 1986, V877, P271 HCAPLUS
(8) Slabas, A; Biochim Biophys Acta 1990, V1039, P181 HCAPLUS
(10) Wallace, A; Protein Eng 1995, V8, P127 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1997:585491 HCAPLUS
DOCUMENT NUMBER: 128:44390
TITLE: Complete genome sequence of Escherichia coli K-12
AUTHOR(S): Blattner, Frederick R.; Plunkett, Guy, III; Bloch, Craig A.; Perna, Nicole T.; Burland, Valerie; Riley, Monica; Collado-Vides, Julio; Glasner, Jeremy D.; Rode, Christopher K.; Mayhew, George F.; Gregor, Jason; Davis, Nelson Wayne; Kirkpatrick, Heather A.; Goeden, Michael A.; Rose, Debra J.; Mau, Bob; Shao, Ying
CORPORATE SOURCE: Lab. Genetics, Univ. Wisconsin-Madison, Madison, WI, 53706, USA
SOURCE: Science (Washington, D. C.) (1997), 277(5331), 1453-1462
CODEN: SCIEAS; ISSN: 0036-8075
PUBLISHER: American Association for the Advancement of Science
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The 4,639,221-base pair sequence of Escherichia coli K-12 is presented. Of 4288 protein-coding genes annotated, 38 percent have no attributed function. Comparison with five other sequenced microbes reveals ubiquitous as well as narrowly distributed gene families; many families of similar genes within E. coli are also evident. The largest family of paralogous proteins contains 80 ABC transporters. The genome as a whole is strikingly organized with respect to the local direction of replication; guanines, oligonucleotides possibly related to replication and recombination, and most genes are so oriented. The genome also contains insertion sequence (IS) elements, phage remnants, and many other patches of unusual compn. indicating genome plasticity through horizontal transfer.

L6 ANSWER 5 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1997:122813 HCAPLUS
DOCUMENT NUMBER: 126:207976
TITLE: A 570-kb DNA sequence of the Escherichia coli K-12 genome corresponding to the 28.0-40.1 min region on the linkage map
AUTHOR(S): Aiba, Hiroji; Baba, Tomoya; Hayashi, Kouji; Inada, Toshifumi; Isono, Katumi; Itoh, Takeshi; Kasai,

Hiroaki; Kashimoto, Kaoru; Kimura, Shigenobu; Kitakawa, Madoka; Kitagawa, Masanari; Makino, Kozo; Miki, Takeyoshi; Mizobuchi, Kiyoshi; Mori, Hirotada; Mori, Tomoko; Motomura, Kouji; Nakade, Shinsuke; Nakamura, Yoshikazu; Nashimoto, Kiroko; Nishio, Yoshitaka; Oshima, Taku; Saito, Noriko; Sampei, Gen-ichi; Seki, Yasushi; Sivasundaram, Suharan; Tagami, Hideaki; Takeda, Jun-ichi; Takemoto, Keiko; Takeuchi, Yasushi; Wada, Chieko; Yamamoto, Yoshihiro; Horiuchi, Takashi

CORPORATE SOURCE: Dep. Molecular Biol., Nagoya Univ., Nagoya, 464-01, Japan

SOURCE: DNA Res. (1996), 3(6), 363-377,435-440
CODEN: DARSE8; ISSN: 1340-2838

PUBLISHER: Kazusa DNA Research Institute

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The contiguous sequence of 569,750 bp corresponding to the region from 28.0 to 40.1 min on the E. coli linkage map was detd. Potential ORFs, RNA-coding genes, and other unique sequences were analyzed by computer.

IT **187857-08-5**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; DNA sequence of 570 kb of the **Escherichia coli** K-12 genome corresponding to the 28.0-40.1 min region on the linkage map)

L6 ANSWER 6 OF 21 HCAPLUS COPYRIGHT 2000 ACS

ACCESSION NUMBER: 1997:55764 HCAPLUS

DOCUMENT NUMBER: 126:248168

TITLE: The enoyl-[acyl-carrier-protein] reductase (FabI) of Escherichia coli, which catalyzes a key regulatory step in fatty acid biosynthesis, accepts NADH and NADPH as cofactors and is inhibited by palmitoyl-CoA

AUTHOR(S): Bergler, Helmut; Fuchsbichler, Sandra; Hoegenauer, Gregor; Turnowsky, Friederike

CORPORATE SOURCE: Institut Mikrobiologie, Universitaet Graz, Graz, A-8010, Austria

SOURCE: Eur. J. Biochem. (1996), 242(3), 689-694
CODEN: EJBCAI; ISSN: 0014-2956

PUBLISHER: Springer

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Redn. of enoyl-acyl-carrier-protein (ACP) substrates by enoyl-ACP reductase is a key regulatory step in fatty acid elongation of E. coli. Two enoyl-ACP reductase activities were described in E. coli, a specific for NADH, the other for NADPH as cofactor. Because of their distinct enzymic properties, these activities were ascribed to 2 different proteins. The NADH-dependent enoyl-ACP reductase of E. coli was identified as the FabI protein, which is the target of a group of antibacterial compds., the diazaborines. We now demonstrate that both enoyl-ACP reductase activities reside in FabI. In crude cell exts. of FabI-overproducing strains, both NADH-dependent and NADPH-dependent enoyl-ACP reductase activities are increased. Mutations in the fabI gene that lead either to temp.-sensitive growth or diazaborine resistance result in the redn. of both activities. When FabI is purified in pH 6.5 buffers, the protein exhibits NADH-dependent and NADPH-dependent reductase activities. Both enzymic activities are inhibited by diazaborine. The NADPH-dependent enoyl-ACP reductase activity, turned out to be approx. 8-fold resistance to diazaborine. The difference in sensitivity indicates that binding of either NADPH or NADH to FabI results in distinct changes in the configuration of the protein or, alternatively, it is different due to the different charge of the cofactors. These effects might be responsible for the differences in the enzymic properties. Both reductase activities of the FabI protein are inhibited by physiol. relevant concns. of palmitoyl-CoA, which might be important in regulating endogenous tatty

acid biosynthesis in E. coli in the presence of exogenous fatty acids.

IT **37251-08-4**
RL: BAC (Biological activity or effector, except adverse); PRP (Properties); BIOL (Biological study)
(enoyl-[acyl-carrier-protein] reductase (FabI) of E. **coli** accepted NADH and NADPH as cofactors and is inhibited by palmitoyl-CoA in fatty acid biosynthesis)

L6 ANSWER 7 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1996:746813 HCAPLUS
DOCUMENT NUMBER: 126:86365
TITLE: Cloning and expression of the multifunctional human fatty acid synthase and its subdomains in Escherichia coli
AUTHOR(S): Jayakumar, Arumugam; Huang, Wei-Yong; Raetz, Beate; Chirala, Subrahmanyam S.; Wakil, Salih J.
CORPORATE SOURCE: Verna Marrs mcLean Dep. Biochem., Baylor Coll. Med., Houston, TX, 77030, USA
SOURCE: Proc. Natl. Acad. Sci. U. S. A. (1996), 93(25), 14509-14514
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We engineered a full-length (8.3-kbp) cDNA coding for fatty acid synthase (FAS; EC 2.3.1.85) from the human brain FAS cDNA clones we characterized previously. In the process of accomplishing this task, we developed a novel PCR procedure, recombinant PCR, which is very useful in joining two overlapping DNA fragments that do not have a common or unique restriction site. The full-length cDNA was cloned in pMAL-c2 for heterologous expression in Escherichia coli as a maltose-binding protein fusion. The recombinant protein was purified by using amylose-resin affinity and hydroxylapatite chromatog. As expected from the coding capacity of the cDNA expressed, the chimeric recombinant protein has a mol. wt. of 310,000 and reacts with antibodies against both human FAS and maltose-binding protein. The maltose-binding protein-human FAS (MBP-hFAS) catalyzed palmitate synthesis from acetyl-CoA, malonyl-CoA, and NADPH and exhibited all of the partial activities of FAS at levels comparable with those of the native human enzyme purified from HepG2 cells. Like the native HepG2 FAS, the products of MBP-hFAS are mainly palmitic acid (>90%) and minimal amts. of stearic and arachidic acids. Similarly, a human FAS cDNA encoding domain 1 (.beta.-ketoacyl synthase, acetyl-CoA and malonyl-CoA transacylases, and .beta.-hydroxyacyl dehydratase) was cloned and expressed in E. coli using pMAL-c2. The expressed fusion protein, MBP-hFAS domain I, was purified to apparent homogeneity (Mr 190,000) and exhibited the activities of the acetyl/malonyl transacylases and the .beta.-hydroxyacyl dehydratase. In addn., a human FAS cDNA encoding domains II and III (enoyl and .beta.-ketoacyl reductases, acyl carrier protein, and thioesterase) was cloned in pET-32b(+) and expressed in E. coli as a fusion protein with thioredoxin and six in-frame histidine residues. The recombinant fusion protein, thioredoxin-human FAS domains II and III, that was purified from E. coli had a mol. wt. of 159,000 and exhibited the activities of the enoyl and .beta.-ketoacyl reductases and the thioesterase. Both the MBP and the thioredoxin-His-tags do not appear to interfere with the catalytic activity of human FAS or its partial activities.

IT **37251-08-4P**, Enoyl [ACP] reductase
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); PRP (Properties); PUR (Purification or recovery); BIOL (Biological study); PREP (Preparation)
(cloning and expression of the multifunctional human fatty acid synthase and its subdomains in **Escherichia coli**)

L6 ANSWER 8 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1996:734567 HCAPLUS
DOCUMENT NUMBER: 126:56745

TITLE: Crystallization of Escherichia coli enoyl reductase and its complex with diazaborine
AUTHOR(S): Baldock, Clair; Rafferty, John B.; Sedelinikova, Svetlana E.; Bithell, Sian; Stuitje, Antoine R.; Slabas, Antoni R.; Rice, David W.
CORPORATE SOURCE: Krebs Inst. Biomolecular Res., Univ. Sheffield, Sheffield, S10 2TN, UK
SOURCE: Acta Crystallogr., Sect. D: Biol. Crystallogr. (1996), D52(6), 1181-1184
CODEN: ABCRE6; ISSN: 0907-4449
PUBLISHER: Munksgaard
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recent work has shown that the NADH-dependent enoyl [acyl carrier protein] reductase (EC 1.3.1.9) (I) from E. coli is the target for diazaborine, an antibacterial agent. Here, I was crystd. by the hanging-drop method of vapor diffusion complexed with NAD and in the presence and absence of a thieno diazaborine. The crystals grown in the absence of diazaborine (form A) were in space group P21 with unit-cell dimensions a = 74.0, b = 81.2, c = 79.0 .ANG., and .beta. = 92.9.degree., and with a tetramer in the asym. unit, whereas those grown in the presence of diazaborine (form B) were in space group P6122 (or P6522) with unit-cell dimensions a = b = 80.9 and c = 328.3 .ANG., and with a dimer in the asym. unit. The structure detn. of this enzyme in the presence of diazaborine will provide information on the nature of the drug binding site and contribute to a program of rational drug design.
IT **37251-08-4**, Enoyl [ACP] reductase **37251-08-4D**, Enoyl [ACP] reductase, complexes with diazaborine deriv.
RL: PRP (Properties)
(crystal structure of **Escherichia coli** enoyl [ACP] reductase and its complex with a diazaborine deriv.)

L6 ANSWER 9 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1996:691493 HCAPLUS
DOCUMENT NUMBER: 125:322752
TITLE: The qmeA (ts) mutation of Escherichia coli is localized in the fabI gene, which encodes enoyl-ACP reductase
AUTHOR(S): Kleerebezem, M.; Heutink, M.; De Cock, H.; Tommassen, J.
CORPORATE SOURCE: Institute Biomembranes, Utrecht University, Utrecht, 3584 CH, Neth.
SOURCE: Res. Microbiol. (1996), 147(8), 609-613
CODEN: RMCREW; ISSN: 0923-2508
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The phenotypes of temp.-sensitive qmeA and fabI mutants of Escherichia coli appear to be very similar. Furthermore, the qmeA mutation could be complemented by the fabI gene on a plasmid, and the fabI allele derived from the qmeA mutant strain harbors a nucleotide substitution identical to that from a previously characterized fabI mutant. These results show that the qmeA gene is, in fact, identical to the fabI gene, which encodes enoyl-ACP reductase, involved in fatty acid elongation.
IT **37251-08-4**, Enoyl-ACP reductase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(qmeA mutation of **Escherichia coli** is localized in fabI gene which encodes enoyl-ACP reductase)

L6 ANSWER 10 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1996:75944 HCAPLUS
DOCUMENT NUMBER: 124:138807
TITLE: Regulation of fatty acid elongation and initiation by acyl-acyl carrier protein in Escherichia coli
AUTHOR(S): Heath, Richard J.; Rock, Charles O.
CORPORATE SOURCE: Dep. of Biochemistry, St. Jude Children's Res. Hospital, Memphis, TN, 38101, USA

SOURCE: J. Biol. Chem. (1996), 271(4), 1833-36
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Long chain acyl-acyl carrier protein (acyl-ACP) has been implicated as a physiol. inhibitor of fatty acid biosynthesis since acyl-ACP degrdn. by thioesterase overexpression leads to constitutive, unregulated fatty acid prodn. The biochem. targets for acyl-ACP inhibition were unknown, and this work identified two biosynthetic enzymes that were sensitive to acyl-ACP feedback inhibition. Palmitoyl-ACP inhibited the incorporation of [14C]malonyl-CoA into long chain fatty acids in cell-free exts. of Escherichia coli. A short chain acyl-ACP species with the electrophoretic properties of .beta.-hydroxybutyryl-ACP accumulated concomitant with the overall decrease in the amt. of [14C]malonyl-CoA incorporation, indicating that the first elongation cycle was targeted by acyl-ACP. All of the proteins required to catalyze the first round of fatty acid synthesis from acetyl-CoA plus malonyl-CoA in vitro were isolated, and the first fatty acid elongation cycle was reconstituted with these purified components. Anal. of the individual enzymes and the pattern of intermediate accumulation in the reconstituted system identified initiation of fatty acid synthesis by .beta.-ketoacyl-ACP synthase III (fabH) and enoyl-ACP reductase (fabI) in the elongation cycle as two steps attenuated by long chain acyl-ACP.

IT **37251-08-4**, Enoyl-ACP reductase
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(feedback inhibition; regulation of fatty acid elongation and initiation by acyl-acyl carrier protein in **Escherichia coli**)

L6 ANSWER 11 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1995:923140 HCAPLUS
DOCUMENT NUMBER: 123:334561
TITLE: Enoyl-acyl carrier protein reductase (fabI) plays a determinant role in completing cycles of fatty acid elongation in Escherichia coli
AUTHOR(S): Heath, Richard J.; Rock, Charles O.
CORPORATE SOURCE: Department Biochemistry, St. Jude Children's Research Hospital, Memphis, TN, 38101, USA
SOURCE: J. Biol. Chem. (1995), 270(44), 26538-42
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The role of enoyl-acyl-carrier protein (ACP) reductase (E.C. 1.3.1.9), the product of the fabI gene, was investigated in the type II, dissocd., fatty acid synthase system of Escherichia coli. All of the proteins required to catalyze one cycle of fatty acid synthesis from acetyl-CoA plus malonyl-CoA to butyryl-ACP in vitro were purified. These proteins were malonyl-CoA:ACP transacylase (fabD), .beta.-ketoacyl-ACP synthase III (fabH), .beta.-ketoacyl-ACP reductase (fabG), .beta.-hydroxydecanoyl-ACP dehydrase (fabA), and enoyl-ACP reductase (fabI). Unlike the other enzymes in the cycle, FabA did not efficiently convert its substrate .beta.-hydroxybutyryl-ACP to crotonyl-ACP, but rather the equil. favored formation of .beta.-hydroxybutyryl-ACP over crotonyl-ACP by a ratio of 9:1. The amt. of butyryl-ACP formed depended on the amt. of FabI protein added to the assay. Exts. from fabI(Ts) mutants accumulated .beta.-hydroxybutyryl-ACP, and the addn. of FabI protein to the fabI(Ts) ext. restored both butyryl-ACP and long-chain acyl-ACP synthesis. FabI was verified to be the only enoyl-ACP reductase required for the synthesis of fatty acids by demonstrating that purified FabI was required for the elongation of both long-chain satd. and unsatd. fatty acids. These results were corroborated by anal. of the intracellular ACP pool compn. in fabI(Ts) mutants that showed .beta.-hydroxybutyryl-ACP and crotonyl-ACP accumulated at the nonpermissive temp. in the same ratio found in the fabI(Ts) exts. and in the in vitro reconstruction expts. that lacked FabI. We conclude that FabI is the only enoyl-ACP reductase involved in fatty acid synthesis in E. coli and that the activity of this enzyme plays a

determinant role in completing cycles of fatty acid biosynthesis.
IT **37251-08-4P**, Enoyl-acyl carrier protein reductase
RL: BAC (Biological activity or effector, except adverse); PUR (Purification or recovery); BIOL (Biological study); PREP (Preparation)
(NADH-dependent; enoyl-acyl-carrier protein reductase (fabI) is a determinant for completion of fatty acid elongation cycles in **Escherichia coli**)

L6 ANSWER 12 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1995:817449 HCAPLUS
DOCUMENT NUMBER: 123:225865
TITLE: Salmonella typhimurium responses to a bactericidal protein from human neutrophils
AUTHOR(S): Qi, Shu-Yun; Szyroki, Alexander; Giles, Ian G.; Moir, Arthur; O'Connor, C. David
CORPORATE SOURCE: Department of Biochemistry, University of Southampton, Southampton, SO16 7PX, UK
SOURCE: Mol. Microbiol. (1995), 17(3), 523-31
CODEN: MOMIEE; ISSN: 0950-382X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Bactericidal/permeability-increasing protein [BPI] is a cationic antimicrobial protein from neutrophils that specifically binds to the surfaces of Gram-neg. bacteria via the lipid A component of lipopolysaccharide. To obtain information about the responses of Salmonella typhimurium to cell-surface damage by BPI, two-dimensional gel electrophoresis and N-terminal microsequencing were used to identify proteins that were induced or repressed following BPI treatment. The majority of the affected proteins are involved in central metabolic processes. Upon addn. of BPI, the .beta.-subunit of the F1 portion of Escherichia coli ATP synthase was repressed threefold whereas six proteins were induced up to 11-fold. Three of the latter were identified as lipoamide dehydrogenase, enoyl-acyl carrier protein reductase, and the heat-shock protein HtpG. Addnl., a novel protein, BipA, was identified that is induced over sevenfold by BPI; sequence anal. suggests that it belongs to the GTPase superfamily and interacts with ribosomes. A conserved direct-repeat motif is present in the regulatory regions of several BPI-inducible genes, including the bipA gene. Only one of the BPI-responsive proteins was induced when cells were treated with polymyxin B, which also binds to lipid A. The authors therefore conclude that BPI and polymyxin B affect different global regulatory networks in S. typhimurium even though they bind with high affinity to the same cell-surface component.
IT **37251-08-4**, Enoyl-acyl carrier protein reductase
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(protein levels of **Salmonella typhimurium** in relation to increased membrane permeability following BPI or polymyxin B treatment)

L6 ANSWER 13 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1995:188664 HCAPLUS
DOCUMENT NUMBER: 122:4340
TITLE: Crystallization and preliminary x-ray diffraction studies of the enoyl-ACP reductase from Escherichia coli
AUTHOR(S): Wagner, Ulrike G.; Bergler, Helmut; Fuchsbichler, Sandra; Turnowsky, Friederike; Hoegenauer, Gregor; Kratky, Christoph
CORPORATE SOURCE: Inst. Physikalische Chemie, Karl-Franzens-Univ. Graz, Graz, A-8010, Austria
SOURCE: J. Mol. Biol. (1994), 243(1), 126-7
CODEN: JMOBAK; ISSN: 0022-2836
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A crystal of the FabI protein [enoyl-acyl-carrier protein (ACP) reductase] from Escherichia coli has been obtained from polyethylene glycol (Mr =

400) soln. with sodium citrate at pH 8.5, by the hanging-drop technique at 4.degree.C. The crystal belongs to the hexagonal space group P6122 (or P6522) with cell dimensions of a = b = 81.1 .ANG. and c = 331.5 .ANG.. There are two mols. in the asym. unit and the crystal diffracts to 2.5 .ANG. resoln.

IT **37251-08-4**, Enoyl-acyl-carrier protein reductase
RL: PEP (Physical, engineering or chemical process); PRP (Properties); PROC (Process)
(crystn. and preliminary x-ray diffraction studies of the enoyl-ACP reductase from **Escherichia coli**)

L6 ANSWER 14 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1995:63837 HCAPLUS
DOCUMENT NUMBER: 122:73533
TITLE: The use of a hybrid genetic system to study the functional relationship between prokaryotic and plant multi-enzyme fatty acid synthetase complexes
AUTHOR(S): Kater, Martin M.; Koningstein, Gregory M.; Nijkamp, H. John J.; Stuitje, Antoine R.
CORPORATE SOURCE: Dep. Genetics, Inst. Molecular Biological Sciences, Amsterdam, 1081 HV, Neth.
SOURCE: Plant Mol. Biol. (1994), 25(5), 771-90
CODEN: PMBIDB; ISSN: 0167-4412
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Fatty acid synthesis in bacteria and plants is catalyzed by a multi-enzyme fatty acid synthetase complex (FAS II) which consists of sep. monofunctional polypeptides. Here the authors present a comparative mol. genetic and biochem. study of the enoyl-ACP reductase FAS components of plant and bacterial origin. The putative bacterial enoyl-ACP reductase gene (envM) was identified on the basis of amino acid sequence similarities with the recently cloned plant enoyl-ACP reductase. Subsequently, it was unambiguously demonstrated by over-expression studies that the envM gene encodes the bacterial enoyl-ACP reductase. An anti-bacterial agent called diazaborine was shown to be a specific inhibitor of the bacterial enoyl-ACP reductase, whereas the plant enzyme was insensitive to this synthetic antibiotic. The close functional relationship between the plant and bacterial enoyl-ACP reductases was inferred from genetic complementation of an envM mutant of Escherichia coli. Ultimately, envM gene-replacement studies, facilitated by the use of diazaborine, demonstrated for the first time that a single component of the plant FAS system can functionally replace its counterpart within the bacterial multienzyme complex. Finally, lipid anal. of recombinant E. coli strains with the hybrid FAS system unexpectedly revealed that enoyl-ACP reductase catalyzes a rate-limiting step in the elongation of unsatd. fatty acids.

IT **148998-18-9**, Protein (**Escherichia coli** clone pHAP1 gene envM reduced)
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); PRP (Properties); BIOL (Biological study); PROC (Process)
(amino acid sequence; functional relationship between prokaryotic and plant multi-enzyme fatty acid synthetase complexes)

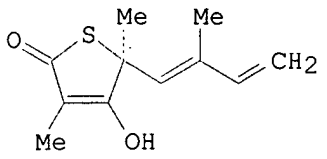
L6 ANSWER 15 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1994:318216 HCAPLUS
DOCUMENT NUMBER: 120:318216
TITLE: Protein EnvM is the NADH-dependent enoyl-ACP reductase (FabI) of Escherichia coli
AUTHOR(S): Bergler, Helmut; Wallner, Petra; Ebeling, Angela; Leitinger, Birgit; Fuchsbichler, Sandra; Aschauer, Heinrich; Kollenz, Gert; Hoegenauer, Gregor; Turnowsky, Friederike
CORPORATE SOURCE: Inst. Mikrobiol., Univ. Graz, Graz, A-8010, Austria
SOURCE: J. Biol. Chem. (1994), 269(8), 5493-6
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal

LANGUAGE: English
AB The EnvM protein was purified from an overproducing Escherichia coli strain. It showed NADH-dependent enoyl-acyl carrier protein (ACP) reductase activity using both crotonyl-ACP and crotonyl-CoA as substrates. The protein bound a radioactive diazaborine deriv. in the presence of NAD+ and radioactive NAD+ in the presence of the drug. Based on these data, it is concluded that EnvM is the NADH-dependent enoyl-ACP reductase (EC 1.3.1.9) of E. coli and the authors propose to rename the corresponding gene fabI.
IT **148998-18-9**, NADH-dependent enoyl-acyl carrier protein reductase (**Escherichia coli** gene fabI) (E.C. 1.3.1.9)
RL: BIOL (Biological study)
(amino acid sequence and activity of and identity of EnvM protein with)
IT **148998-19-0**, NADH-dependent enoyl-acyl carrier protein reductase variant (**Escherichia coli** gene fabI diazaborine resistance-conferring) **148998-20-3**, NADH-dependent enoyl-acyl carrier protein reductase variant (**Escherichia coli** JP1111 temp.-sensitive mutant gene fabI)
RL: PRP (Properties); BIOL (Biological study)
(amino acid sequence of)

L6 ANSWER 16 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1990:192764 HCAPLUS
DOCUMENT NUMBER: 112:192764
TITLE: envM genes of Salmonella typhimurium and Escherichia coli
AUTHOR(S): Turnowsky, Friederike; Fuchs, Karoline; Jeschek, Claudia; Hoegenauer, Gregor
CORPORATE SOURCE: Inst. Mikrobiol., Univ. Graz, Graz, A-8010, Austria
SOURCE: J. Bacteriol. (1989), 171(12), 6555-65
CODEN: JOBAAY; ISSN: 0021-9193
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Conjugation and bacteriophage P1 transduction expts. in E. coli showed that resistance to the antibacterial compd. diazaborine is caused by an allelic form of the envM gene. The envM gene from S. typhimurium was cloned and sequenced. It codes for a 27,765-dalton protein. The plasmids carrying this DNA complemented a conditionally lethal envM mutant of E. coli. Recombinant plasmids contg. gene envM from a diazaborine-resistant S. typhimurium strain conferred the drug resistance phenotype to susceptible E. coli cells. A guanine-to-adenine exchange in the envM gene changing a Gly codon to a Ser codon was shown to be responsible for the resistance character. Upstream of envM a small gene coding for a 10,445-dalton protein was identified. Incubating a temp.-sensitive E. coli envM mutant at the nonpermissive temp. caused effects on the cells similar to those caused by treatment with diazaborine, i.e., inhibition of fatty acid, phospholipid, and lipopolysaccharide biosynthesis, induction of a 28,000-dalton inner membrane protein, and change in the ratio of the porins OmpC and OmpF.
IT **126731-07-5**, Protein (**Salmonella typhimurium** clone pFT501 gene envM reduced) **126731-08-6**, Protein (**Salmonella typhimurium** clone pKF403 gene envM reduced)
RL: PRP (Properties)
(amino acid sequence of)
IT **126730-36-7**, Deoxyribonucleic acid (**Salmonella typhimurium** clone pFT501 gene envM) **126730-38-9**, Deoxyribonucleic acid (**Salmonella typhimurium** clone pKF403 gene envM)
RL: PRP (Properties); BIOL (Biological study)
(nucleotide sequence of)

L6 ANSWER 17 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1986:606013 HCAPLUS
DOCUMENT NUMBER: 105:206013
TITLE: Effect of thiolactomycin on the individual enzymes of the fatty acid synthase system in Escherichia coli

AUTHOR(S): Nishida, Ikuo; Kawaguchi, Akihiko; Yamada, Mitsuhiro
CORPORATE SOURCE: Dep. Biol., Univ. Tokyo, Tokyo, 153, Japan
SOURCE: J. Biochem. (Tokyo) (1986), 99(5), 1447-54
CODEN: JOBIAO; ISSN: 0021-924X
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



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AB Thiolactomycin (I) selectively inhibits type II fatty acid synthases. The mode of I action on the fatty acid synthase system of E. coli was investigated. Of the 6 individual enzymes of the fatty acid synthase system, [acyl-carrier-protein] (ACP) acetyltransferase and 3-oxoacyl-ACP synthase were inhibited by I. The other enzymes were not affected by this antibiotic. Inhibition of the fatty acid synthase system by I was reversible. As to ACP acetyltransferase, the inhibition was competitive with respect to ACP and noncompetitive with respect to acetyl-CoA. As to 3-oxoacyl-ACP synthase, the inhibition was competitive with respect to malonyl-ACP and noncompetitive with respect to acetyl-ACP. I action on the fatty acid synthase system was comparable with that of cerulenin.

IT **37251-08-4**
RL: PROC (Process)
(inhibition of, of **Escherichia coli** by thiolactomycin)

L6 ANSWER 18 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1986:123250 HCAPLUS
DOCUMENT NUMBER: 104:123250
TITLE: Analytical problems with the determination and interpretation of the testosterone-epitestosterone quotient within the scope of doping controls
AUTHOR(S): Clausnitzer, C.; Grosse, J.
CORPORATE SOURCE: Zentralinst. Sportmed. Dienstes, Kreischa, DDR-8216, Ger. Dem. Rep.
SOURCE: Med. Sport (Berlin) (1985), 25(7), 212-15
CODEN: MESPBQ; ISSN: 0025-8415
DOCUMENT TYPE: Journal
LANGUAGE: German

AB Anal. of the urine of athletes for testosterone [58-22-0] and epitestosterone [481-30-1], whose ratio provides an indication of exogenous testosterones use, requires the hydrolysis of steroid conjugates prior to gas chromatog.-mass spectrometry. The commonly used .beta.-glucuronidase [9001-45-0] prepn. from Helix pomatia may give falsely high testosterone values, however, because this prepn. also contains enzymes that convert androstenediol [**521-17-5**] to testosterone. This interference can be avoided by using .beta.-glucuronidase from **Escherichia coli**, since it does not produce the undesired side reactions. Consequently, the latter enzyme is recommended for urine analyses designed to detect improper use of testosterone by athletes.

L6 ANSWER 19 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1984:505135 HCAPLUS
DOCUMENT NUMBER: 101:105135
TITLE: Molecular cloning of the yeast fatty acid synthetase genes, FAS1 and FAS2: illustrating the structure of the FAS1 cluster gene by transcript mapping and

transformation studies
AUTHOR(S): Schweizer, Michael; Lebert, Cordula; Hoeltke, Joachim; Roberts, Lilian M.; Schweizer, Eckhart
CORPORATE SOURCE: Univ. Erlangen-Nuernberg, Erlangen, D-8520, Fed. Rep. Ger.
SOURCE: MGG, Mol. Gen. Genet. (1984), 194(3), 457-65
CODEN: MGGEAE; ISSN: 0026-8925
DOCUMENT TYPE: Journal
LANGUAGE: English

AB From a Saccharomyces cerevisiae gene bank in the novel yeast cosmid shuttle vector pMS201, the fatty acid synthetase (FAS) [9045-77-6] genes FAW1 and FAS2 were isolated. FAS clones were identified by in situ colony hybridization with 2 yeast DNA probes apparently capable of producing avian FAS cross-reacting material. Classification as FAS1 or FAS2 clones was achieved by specific transformation of fas1 and fas2 yeast mutants. By transcription mapping, FAS1 was assigned to .apprx.5.3 kilobases (kb) within 14.8 kb of chromosomal DNA covered by 2 genomically adjacent BamHI fragments. The FAS2 gene was located on a single BamHI fragment of 25 kb. One of the FAS clones (FAS2) produces immunol. cross-reacting material in **Escherichia coli**. High-frequency transformation of fas1 mutants was only obsd. with 1 subclone, pMS3021, contg. the intact FAS1 locus. Other DNA segments cloned in the same self-replicating vector but representing only part of FAS1 exhibited drastically lower transformation rates. As evident from this and from FAS1-TRP1-cotransformation rates, only the intact FAS1 gene in pMS3021 is capable of fas1-mutant complementation. With partial FAS1 genes, even when they code for an intact equiv. of the mutated domain, chromosomal integration is necessary for the expression of FAS. In integrative transformants, the coexistence of integrated and autonomously replicating plasmid DNA was demonstrated. Both the extrachromosomal and chromosomally integrated FAS DNA was mitotically unstable. Transformation studies with subcloned FAS1 DNA segments revealed the relative locations of the enol reductase [**37251-08-4**] and dehydratase [9027-13-8] domains within this pentafunctional cluster gene.

L6 ANSWER 20 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1981:457130 HCAPLUS
DOCUMENT NUMBER: 95:57130
TITLE: Steric course of reaction catalyzed by the enoyl acyl-carrier-protein reductase of Escherichia coli
AUTHOR(S): Saito, Kazuki; Kawaguchi, Akihiko; Seyama, Yousuke; Yamakawa, Tamio; Okuda, Shigenobu
CORPORATE SOURCE: Inst. Appl. Microbiol., Univ. Tokyo, Tokyo, 113, Japan
SOURCE: Eur. J. Biochem. (1981), 116(3), 581-6
CODEN: EJBCAI; ISSN: 0014-2956
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The steric course of the reaction catalyzed by E. coli enoyl-[acyl-carrier-protein] reductase (I) was studied. trans-2-[2-2H1decenoic and trans-2-[3-2H1]decenoic acids were synthesized and converted to the corresponding decenoyl thiol esters with CoA or acyl carrier protein. These 2H-labeled decenoyl thiol esters were incubated with purified I in the presence of NADPH or NADH. The unlabeled trans-2-decenoyl thiol esters were incubated with I in the presence of (4S)-[4-2H1]NADH. The unlabeled decenoyl thiol esters were also incubated with the enzyme in 2H2O. The decanoic acids formed in the above incubations were extd. and subjected to the action of acyl-CoA oxidase, which had been previously shown to catalyze the anti elimination of the pro-2R and pro-3R H atoms of acyl-CoA. The resulting products, 2-decenoyl-CoAs, were converted to Me esters and their 2H contents were analyzed by gas chromatog./mass spectrometry. The results suggested that the redn. catalyzed by E. coli I occurs by a syn addn. of H via a 2-Re, 3-Si attack on the double bond.

IT **37251-08-4**
RL: MSC (Miscellaneous); PRP (Properties)
(reaction mechanism of, of **Escherichia coli**,

stereochem. of)

L6 ANSWER 21 OF 21 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1972:548655 HCAPLUS
DOCUMENT NUMBER: 77:148655
TITLE: Acyl carrier protein. XVI. Intermediate reactions of unsaturated fatty acid synthesis in Escherichia coli and studies of fab B mutants
AUTHOR(S): Birge, Claire H.; Vagelos, P. Roy
CORPORATE SOURCE: Sch. Med., Washington Univ., St. Louis, Mo., USA
SOURCE: J. Biol. Chem. (1972), 247(16), 4921-9
CODEN: JBCHA3
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Synthesis of cis-5-.beta.-hydroxyl-[2-14C]-dodecenoyl-acyl carrier protein (ACP), a postulated intermediate in unsatd. fatty acid synthesis in E. coli, was achieved from cis-3-decenoyl-ACP and [2-14C]-malonyl-ACP through the combined action of two enzymes, .beta.-ketoacyl-ACP synthetase and .beta.-keto-acyl-ACP reductase. The conversion of cis-5-.beta.-hydroxyl-[2-14C]-dodecenoyl-ACP to cis-5-trans-2-[2-14C]-dodecadienoyl-ACP and to cis-5-[2-14C]-dodecenoyl-ACP was shown utilizing a crude fattv acid synthetase prepn. of wild type E. coli. Elongation of cis-5-.beta.-hydroxyl-[2-14C]-dodecenoyl-ACP to long-chain unsatd. fatty acids was also shown with this fatty acid synthetase prepn. These studies indicate that cis-5-.beta.-hydroxydodecenoyl-ACP, cis-5-trans-2-dodecadienoyl-ACP, and cis-5-dodecenoyl-ACP are intermediates in the synthesis of long-chain unsatd. fatty acids. Exts. of a class of fatty acid biosynthetic mutants, fab B, which specifically cannot synthesize unsatd. fatty acids although synthesizing satd. fatty acids normally, were examd. for all the above activities. No apparent defect was revealed in any step involved in the utilization of cis-5-.beta.-hydroxy-[2-14C]-dodecenoyl-ACP for long-chain unsatd. fatty acid synthesis. Mutant exts. were also found to contain normal amts. of .beta.-ketoacyl-ACP reductase and .beta.-hydroxyacyl-ACP dehydrase, which apparently function in both the satd. and unsatd. fatty acid biosynthetic pathways, and of enoyl-ACP reductase. It is concluded that fab B mutants contain a mutation in an enzyme which catalyzes a reaction unique to the unsatd. fatty acid pathway which is yetunknown.
IT **37251-08-4**
RL: BIOL (Biological study)
(in unsatd. fatty acids formation, by **Escherichia coli**)

=> D STAT QUE L12

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON 521-17-5/RN
L2 12 SEA FILE=REGISTRY ABB=ON PLU=ON ENVM/BI
L3 39 SEA FILE=REGISTRY ABB=ON PLU=ON FABI/BI
L4 13 SEA FILE=REGISTRY ABB=ON PLU=ON (ENOYL-/CN OR "ENOYL-(ACYL CARRIER PROTEIN) REDUCTASE"/CN OR "ENOYL-(ACYL-CARRIER PROTEIN) REDUCTASE (CHLAMYDIA MURIDARUM STRAIN NIGG GENE TC0380)"/CN OR "ENOYL-(ACYL-CARRIER PROTEIN) REDUCTASE (CHLAMYDOPHILA PNEUMONIAE AR39 STRAIN AR39 GENE CP0349)"/CN OR "ENOYL-(ACYL-CARRIER PROTEIN) REDUCTASE (PSEUDOMONAS AERUGINOSA STRAIN PAO1 GENE FABI)"/CN OR "ENOYL-(ACYL-CARRIER-PROT EIN) REDUCTASE (NADH) (BUCHNERA STRAIN APS GENE FABI)"/CN OR "ENOYL-(ACYL-CARRIER-PROTEIN) REDUCTASE (NADH) (AQUIFEX AEOLICUS GENE FABI)"/CN OR "ENOYL-(ACYL-CARRIER-PROTEIN) REDUCTASE (NADH) (CAMPYLOBACTER JEJUNI STRAIN NCTC 11168 GENE FABI)"/CN OR "ENOYL-(ACYL-CARRIER-PROTEIN) REDUCTASE (NEISSERIA MENINGITIDIS STRAIN MD58 GENE NMB0336)"/CN OR "ENOYL-ACP REDUCTASE"/CN OR "ENOYL-ACP REDUCTASE (ENR-A) (ARABIDOPSIS THALIANA GENE AT2G05990)"/CN OR "ENOYL-ACP REDUCTASE (ESCHERICH IA COLI CLONE PEAR3 GENE ENVM)"/CN OR "ENOYL-ACP REDUCTASE (NEISSERIA MENINGITIDIS STRAIN Z2491 GENE FABI)"/CN OR

"ENOYL-ACYL CARRIER PROTEIN REDUCTASE (DEINOCOCCUS RADIODURANS STRAIN R1 GENE DR1967)"/CN OR "ENOYL-ACYL-CARRIER PROTEIN REDUCTASE (CHLAMYDIA PNEUMONIAE GENE FABI)"/CN OR "ENOYL-ACYL-CARRIER PROTEIN REDUCTASE (CHLAMYDIA TRACHOMATIS GENE FABI)"/CN)

L5 1169 SEA FILE=HCAPLUS ABB=ON PLU=ON L1 OR L2 OR L3 OR L4
L6 21 SEA FILE=HCAPLUS ABB=ON PLU=ON L5(L)(ESCH? OR COLI OR SALMONEL? OR TYPHIM?)
L11 131 SEA FILE=HCAPLUS ABB=ON PLU=ON ENVM OR ENV(W)M OR ENOYL?(2A)(ACP OR ACYL(W)CARRIER(W)PROTEIN)(2A)REDUCTASE
L12 40 SEA FILE=HCAPLUS ABB=ON PLU=ON (L11(L)(ESCH? OR COLI OR SALMONEL? OR TYPHIM?)) NOT L6

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=> D IBIB ABS HITRN L12 1-40

L12 ANSWER 1 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 2000:540430 HCAPLUS
DOCUMENT NUMBER: 133:220072
TITLE: Microbiology: A triclosan-resistant bacterial enzyme
AUTHOR(S): Heath, Richard J.; Rock, Charles O.
CORPORATE SOURCE: Dep. Biochem., St Jude Children's Res. Hosp., Memphis, TN, 38105, USA
SOURCE: Nature (London) (2000), 406(6792), 145-146
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Triclosan is an antimicrobial agent that acts by inhibiting enoyl-ACP-reductase (FabI) in many microorganisms. The isolation and characterization of a unique triclosan resistant enoyl-ACP-reductase from Streptococcus pneumoniae (FabK) is reported. The purified FabK protein is a 34 kd protein that contains 0.8 mols. of FAD per FabK monomer and requires NADH for activity. The FabK protein is 100 fold more triclosan resistant than the FabI protein. The implications of triclosan resistant enoyl-ACP-reductase to new drug development are discussed.
REFERENCE COUNT: 21
REFERENCE(S): (1) Azuma, R; J Chem Phys 1999, V111, P8666 HCAPLUS
(2) Berland, K; Biophys J 1995, V68, P694 HCAPLUS
(6) Heath, R; J Biol Chem 1996, V271, P1833 HCAPLUS
(7) Heath, R; J Biol Chem 1998, V273, P30316 HCAPLUS
(8) Heath, R; J Biol Chem 1999, V274, P11110 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 2000:380165 HCAPLUS
DOCUMENT NUMBER: 133:161156
TITLE: Inhibition of InhA, the Enoyl Reductase from Mycobacterium tuberculosis, by Triclosan and Isoniazid
AUTHOR(S): Parikh, Sapan L.; Xiao, Guoping; Tonge, Peter J.
CORPORATE SOURCE: Department of Chemistry and Graduate Programs in Biophysics and Molecular and Cellular Biochemistry, State University of New York at Stony Brook, Stony Brook, NY, 11794-3400, USA
SOURCE: Biochemistry (2000), 39(26), 7645-7650
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Structural and genetic studies indicate that the antibacterial compd. triclosan, an additive in many personal care products, is an inhibitor of

EnvM, the enoyl reductase from **Escherichia coli**
. Here we show that triclosan specifically inhibits InhA, the enoyl reductase from Mycobacterium tuberculosis and a target for the antitubercular drug isoniazid. Binding of triclosan to wild-type InhA is uncompetitive with respect to both NADH and trans-2-dodecenoyl-CoA, with Ki' values of 0.22 .+-. 0.02 and 0.21 .+-. 0.01 .mu.M, resp. Replacement of Y158, the catalytic tyrosine residue, with Phe, reduces the affinity of triclosan for the enzyme and results in noncompetitive inhibition, with Ki and Ki' values of 36 .+-. 5 and 47 .+-. 5 .mu.M, resp. Consequently, the Y158 hydroxyl group is important for triclosan binding, suggesting that triclosan binds in similar ways to both InhA and **EnvM**. In addn., the M161V and A124V InhA mutants, which result in resistance of Mycobacterium smegmatis to triclosan, show significantly reduced affinity for triclosan. Inhibition of M161V is noncompetitive with Ki' = 4.3 .+-. 0.5 .mu.M and Ki = 4.4 .+-. 0.9 .mu.M, while inhibition of A124V is uncompetitive with Ki' = 0.81 .+-. 0.11 .mu.M. These data support the hypothesis that the mycobacterial enoyl reductases are targets for triclosan. The M161V and A124V enzymes are also much less sensitive to isoniazid compared to the wild-type enzyme, indicating that triclosan can stimulate the emergence of isoniazid-resistant enoyl reductases. In contrast, I47T and I21V, two InhA mutations that occur in isoniazid-resistant clin. isolates of M. tuberculosis, show unimpaired inhibition by triclosan, with uncompetitive inhibition consts. (Ki') of 0.18 .+-. 0.01 and 0.12 .+-. 0.01 .mu.M, resp. The latter result indicates that InhA inhibitors targeted at the enoyl substrate binding site may be effective against existing isoniazid-resistant strains of M. tuberculosis.

REFERENCE COUNT: 27
REFERENCE(S):
(1) Banerjee, A; Science 1994, V263, P227 HCAPLUS
(2) Basso, L; J Infect Dis 1998, V178, P769 HCAPLUS
(3) Blanchard, J; Annu Rev Biochem 1996, V65, P215 HCAPLUS
(5) Dessen, A; Science 1995, V267, P1638 HCAPLUS
(6) Greenman, J; J Antimicrob Chemother 1997, V40, P659 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 2000:148483 HCAPLUS
DOCUMENT NUMBER: 132:276483
TITLE: Inhibition of the Staphylococcus aureus NADPH-dependent enoyl-acyl carrier protein reductase by triclosan and hexachlorophene
AUTHOR(S): Heath, Richard J.; Li, Jing; Roland, Gregory E.; Rock, Charles O.
CORPORATE SOURCE: Department of Biochemistry, St. Jude Children's Research Hospital, Memphis, TN, 38105, USA
SOURCE: J. Biol. Chem. (2000), 275(7), 4654-4659
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Enoyl-acyl carrier protein reductase** (FabI) plays a determinant role in completing cycles of elongation in type II fatty acid synthase systems and is an important target for antibacterial drugs. The FabI component of Staphylococcus aureus (saFabI) was identified, and its properties were compared with **Escherichia coli** FabI (ecFabI). The ecFabI and saFabI had similar specific activities, and saFabI expression complemented the E. **coli** fabI(Ts) mutant, illustrating that the Gram-pos. FabI was interchangeable with the Gram-neg. FabI enzyme. However, ecFabI was specific for NADH, whereas saFabI exhibited specific and pos. cooperative binding of NADPH. Triclosan and hexachlorophene inhibited both ecFabI and saFabI. The triclosan-resistant ecFabI(G93V) protein was also refractory to hexachlorophene inhibition, illustrating that both drugs bind at the

FabI active site. Both the introduction of a plasmid expressing the safabI gene or a missense mutation in the chromosomal safabI gene led to triclosan resistance in S. aureus; however, these strains did not exhibit cross-resistance to hexachlorophene. The replacement of the ether linkage in triclosan by a carbon bridge in hexachlorophene prevented the formation of a stable FabI-NAD(P)+-drug ternary complex. Thus, the formation of this ternary complex is a key determinant of the antibacterial activity of FabI inhibitors.

REFERENCE COUNT: 32
REFERENCE(S):
(1) Baldock, C; Biochem Pharmacol 1998, V55, P1541 HCAPLUS
(2) Baldock, C; Science 1996, V274, P2107 HCAPLUS
(3) Banerjee, A; Science 1994, V263, P227 HCAPLUS
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(5) Bergler, H; J Biol Chem 1994, V269, P5493 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 2000:82159 HCAPLUS
DOCUMENT NUMBER: 132:134077
TITLE: Crystallization of the NADP-dependent .beta.-keto acyl-carrier protein reductase from Brassica napus
AUTHOR(S): Fisher, Martin; Sedelnikova, Svetlana E.; Martindale, Wayne; Thomas, Neil C.; Simon, J. William; Slabas, Antoni R.; Rafferty, John B.
CORPORATE SOURCE: Krebs Institute for Biomolecular Research, Department of Molecular Biology and Biotechnology, The University of Sheffield, Sheffield, S10 2TN, UK
SOURCE: Acta Crystallogr., Sect. D: Biol. Crystallogr. (2000), D56(1), 86-88
CODEN: ABCRE6; ISSN: 0907-4449
PUBLISHER: Munksgaard International Publishers Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB NADP-dependent .beta.-ketoacyl-[acyl carrier protein (ACP)] reductase (I) from B. napus was crystd. by the hanging-drop vapor-diffusion method using polyethylene glycol of av. mol. wt. 1500 as the precipitant. The crystals belonged to the hexagonal space group P6422, with unit-cell parameters a = b = 129.9, c = 93.1 .ANG., and .alpha. = .beta. = 90 and .gamma. = 120.degree.. Calcd. values for Vm, the use of rotation and translation functions, and consideration of the packing suggested that the asym. unit contained a monomer. The crystals diffracted to beyond 2.8 .ANG. resoln. and were more amenable to x-ray diffraction anal. than those reported previously for the **Escherichia coli** enzyme. The structure detn. of B. napus I will provide important insights into the catalytic mechanism of the enzyme and into the evolution of the fatty acid elongation cycle by comparisons with the other oxidoreductase of the pathway, **enoyl-[ACP] reductase**.

REFERENCE COUNT: 13
REFERENCE(S):
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1999:700572 HCAPLUS
DOCUMENT NUMBER: 132:32628
TITLE: Inhibitor binding studies on enoyl reductase reveal conformational changes related to substrate recognition
AUTHOR(S): Roujeinikova, Anna; Sedelnikova, Svetlana; De Boer, Gert-Jan; Stuitje, Antoine R.; Slabas, Antoni R.;

Rafferty, John B.; Rice, David W.
CORPORATE SOURCE: Krebs Institute for Biomolecular Research, Department of Molecular Biology and Biotechnology, University of Sheffield, Sheffield, S10 2TN, UK
SOURCE: J. Biol. Chem. (1999), 274(43), 30811-30817
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Enoyl acyl carrier protein reductase** (ENR) is involved in fatty acid biosynthesis. In **Escherichia coli** this enzyme is the target for the exptl. family of antibacterial agents, the diazaborines, and for triclosan, a broad spectrum antimicrobial agent. Biochem. studies have suggested that the mechanism of diazaborine inhibition is dependent on NAD+ and not NADH, and resistance of Brassica napus ENR to diazaborines is thought to be due to the replacement of a glycine in the active site of the E. **coli** enzyme by an alanine at position 138 in the plant homolog. We present here an x-ray anal. of crystals of B. napus ENR A138G grown in the presence of either NAD+ or NADH and the structures of the corresponding ternary complexes with thienodiazaborine obtained either by soaking the drug into the crystals or by co-crystn. of the mutant with NAD+ and diazaborine. Anal. of the ENR A138G complex with diazaborine and NAD+ shows that the site of diazaborine binding is remarkably close to that reported for E. **coli** ENR. However, the structure of the ternary ENR A138G-NAD+-diazaborine complex obtained using co-crystn. reveals a previously unobserved conformational change affecting 11 residues that flank the active site and move closer to the nicotinamide moiety making extensive van der Waals contacts with diazaborine. Considerations of the mode of substrate binding suggest that this conformational change may reflect a structure of ENR that is important in catalysis.
REFERENCE COUNT: 26
REFERENCE(S): (2) Baldock, C; J Mol Biol 1998, V284, P1529 HCAPLUS
(3) Baldock, C; Science 1996, V274, P2107 HCAPLUS
(4) Bergler, H; J Biol Chem 1994, V269, P5493 HCAPLUS
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 6 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1999:581656 HCAPLUS
DOCUMENT NUMBER: 132:19316
TITLE: Construction and use of low-copy number T7 expression vectors for purification of problem proteins: purification of Mycobacterium tuberculosis RmlD and Pseudomonas aeruginosa LasI and RhlI proteins, and functional analysis of purified RhlI
AUTHOR(S): Hoang, T. T.; Ma, Y.; Stern, R. J.; McNeil, M. R.; Schweizer, H. P.
CORPORATE SOURCE: Department of Microbiology, Colorado State University, Fort Collins, CO, USA
SOURCE: Gene (1999), 237(2), 361-371
CODEN: GENED6; ISSN: 0378-1119
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Purifn. of proteins from **Escherichia coli** under native conditions is often hampered by inclusion-body formation after overexpression from T7 promoter-based expression vectors. This is probably due to the relatively high copy no. of the ColE1-based expression vectors. To circumvent these problems, the low-copy-no. pViet and pNam expression vectors were constructed. These vectors contain the pSC101 origin of replication and allow the expression of oligohistidine and intein chitin-binding domain fusion proteins, resp. Since pViet and pNam

do not replicate in E. **coli** B strains, an E. **coli** K-12 host strain [SA1503(DE3)] was constructed. This strain is defective in the Lon and OmpT proteases and allows IPTG-inducible expression of recombinant proteins from the T7 promoter. The new vectors were successfully tested by purifn. of three very insol. proteins (RmlD, LasI and RhlI) under non-denaturing conditions, and all three proteins retained enzymic activity. The purified hexahistidine (His6)-tagged Pseudomonas aeruginosa RhlI protein was subjected to more detailed analyses, which indicated that (1) only butyryl-acyl carrier protein (ACP) and S-adenosylmethionine (SAM) were required for synthesis of N-butyryl-l-homoserine lactone; (2) when present at physiol. concns., butyryl-CoA and NADPH were not substrates for RhlI; (3) RhlI was able to synthesize N-hexanoyl-l-homoserine lactone from hexanoyl-ACP and SAM; (4) RhlI was able to direct synthesis of N-butyryl-l-homoserine lactone from crotonyl-ACP in a reaction coupled to purified P. aeruginosa FabI (**enoyl-ACP reductase**).

REFERENCE COUNT: 38

REFERENCE(S):
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(2) Belisle, J; J Bacteriol 1991, V173, P6991 HCAPLUS
(3) Bergler, H; J Biol Chem 1994, V269, P5493 HCAPLUS
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(5) Chen, H; Analyt Biochem 1993, V212, P295 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 7 OF 40 HCAPLUS COPYRIGHT 2000 ACS

ACCESSION NUMBER: 1999:539038 HCAPLUS
DOCUMENT NUMBER: 131:295159

TITLE: Kinetic and Structural Characteristics of the Inhibition of Enoyl (Acyl Carrier Protein) Reductase by Triclosan

AUTHOR(S): Ward, Walter H. J.; Holdgate, Geoffrey A.; Rowsell, Sian; McLean, Estelle G.; Pauptit, Richard A.; Clayton, Edward; Nichols, Wright W.; Colls, Jeremy G.; Minshull, Claire A.; Jude, David A.; Mistry, Anil; Timms, David; Camble, Roger; Hales, Neil J.; Britton, Carolyn J.; Taylor, Ian W. F.
CORPORATE SOURCE: AstraZeneca, Mereside Alderley Park Macclesfield Cheshire, SK10 4TG, UK

SOURCE: Biochemistry (1999), 38(38), 12514-12525
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Triclosan is used widely as an antibacterial agent in dermatol. products, mouthwashes, and toothpastes. Recent studies imply that antibacterial activity results from binding to **enoyl** (**acyl carrier protein**) **reductase** (EACPR, EC 1.3.1.9). We first recognized the ability of triclosan to inhibit EACPR from **Escherichia coli** in a high throughput screen where the enzyme and test compd. were preincubated with NAD+, which is a product of the reaction. The concn. of triclosan required for 50% inhibition approximates to 50% of the enzyme concn., indicating that the free compd. is depleted by binding to EACPR. With no preincubation or added NAD+, the degree of inhibition by 150 nM triclosan increases gradually over several minutes. The onset of inhibition is more rapid when NAD+ is added. Gel filtration and mass spectrometry show that inhibition by triclosan is reversible. Steady-state assays were designed to avoid depletion of free inhibitor and changes in the degree of inhibition. The results suggest that triclosan binds to E-NAD+ complex, with a dissocn. const. around 20-40 pM. Triclosan follows competitive kinetics with respect to NADH, giving an inhibition const. of 38 pM at zero NADH and satg. NAD+. Uncompetitive kinetics are obsd. when NAD+ is varied, giving an inhibition const. of 22 pM at satg. NAD+. By following regain of catalytic activity after diln. of EACPR that had been preincubated with triclosan and NAD+, the rate const. for dissocn. of the

inhibitor (koff) is measured as 1.9.times.10-4 s-1. The assocn. rate const. (kon) is estd. as 2.6.times.107 s-1 M-1 by monitoring the onset of inhibition during assays started by addn. of EACPR. As expected, the ratio koff/kon = 7.1 pM is similar to the inhibition consts. from the steady-state studies. The crystal structure of E. **coli** EACPR in a complex with coenzyme and triclosan has been detd. at 1.9 .ANG. resoln., showing that this compd. binds in a similar site to the diazaborine inhibitors. The high affinity of triclosan appears to be due to structural similarity to a tightly bound intermediate in catalysis.

REFERENCE COUNT: 24
REFERENCE(S):
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 8 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1999:427517 HCAPLUS
DOCUMENT NUMBER: 131:252126
TITLE: Structural Basis and Mechanism of Enoyl Reductase Inhibition by Triclosan
AUTHOR(S): Stewart, Michael J.; Parikh, Sapan; Xiao, Guoping; Tonge, Peter J.; Kisker, Caroline
CORPORATE SOURCE: Department of Pharmacological Sciences, SUNY at Stony Brook, Stony Brook, NY, 11794-8651, USA
SOURCE: J. Mol. Biol. (1999), 290(4), 859-865
CODEN: JMOBAK; ISSN: 0022-2836
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The **enoyl-acyl carrier protein reductase** (ENR) is involved in bacterial fatty acid biosynthesis and is the target of the antibacterial diazaborine compds. and the front-line antituberculosis drug isoniazid. Recent studies suggest that ENR is also the target for the broad-spectrum biocide triclosan. The 1.75 .ANG. crystal structure of **EnvM**, the ENR from **Escherichia coli**, in complex with triclosan and NADH reveals that triclosan binds specifically to **EnvM**. These data provide a mol. mechanism for the antibacterial activity of triclosan and substantiate the hypothesis that its activity results from inhibition of a specific cellular target rather than non-specific disruption of the bacterial cell membrane. This has important implications for the emergence of drug-resistant bacteria, since triclosan is an additive in many personal care products such as toothpastes, mouthwashes and soaps. Based on this structure, rational design of triclosan derivs. is possible which might be effective against recently identified triclosan-resistant bacterial strains. (c) 1999 Academic Press.

REFERENCE COUNT: 18
REFERENCE(S):
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 9 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1999:265529 HCAPLUS
DOCUMENT NUMBER: 131:67686
TITLE: Mechanism of triclosan inhibition of bacterial fatty acid synthesis
AUTHOR(S): Heath, Richard J.; Rubin, J. Ronald; Holland, Debra R.; Zhang, Erli; Snow, Mark. E.; Rock, Charles O.
CORPORATE SOURCE: Department of Biochemistry, St. Jude Children's

Research Hospital, Memphis, TN, 38105, USA
SOURCE: J. Biol. Chem. (1999), 274(16), 11110-11114
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Triclosan is a broad-spectrum antibacterial agent that inhibits bacterial fatty acid synthesis at the **enoyl-acyl carrier protein reductase** (FabI) step. Resistance to triclosan in **Escherichia coli** is acquired through a missense mutation in the fabI gene that leads to the expression of FabI[G93V]. The specific activity and substrate affinities of FabI[G93V] are similar to FabI. Two different binding assays establish that triclosan dramatically increases the affinity of FabI for NAD+. In contrast, triclosan does not increase the binding of NAD+ to FabI[G93V]. The x-ray crystal structure of the FabI-NAD+-triclosan complex confirms that hydrogen bonds and hydrophobic interactions between triclosan and both the protein and the NAD+ cofactor contribute to the formation of a stable ternary complex, with the drug binding at the enoyl substrate site. These data show that the formation of a noncovalent "bi-substrate" complex accounts for the effectiveness of triclosan as a FabI inhibitor and illustrates that mutations in the FabI active site that interfere with the formation of a stable FabI-NAD+-triclosan ternary complex acquire resistance to the drug.

REFERENCE COUNT: 27
REFERENCE(S):
(1) Baldock, C; Science 1996, V274, P2107 HCAPLUS
(2) Banerjee, A; Science 1994, V263, P227 HCAPLUS
(4) Bergler, H; Eur J Biochem 1996, V242, P689 HCAPLUS
(5) Bergler, H; J Biol Chem 1994, V269, P5493 HCAPLUS
(6) Bergler, H; J Gen Microbiol 1992, V138, P2093 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 10 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1999:216800 HCAPLUS
DOCUMENT NUMBER: 131:83752
TITLE: Genes coding for phosphotransacetylase and acetate kinase in Sinorhizobium meliloti are in an operon that is inducible by phosphate stress and controlled by PhoB
AUTHOR(S): Summers, Michael L.; Denton, Michael C.; McDermott, Timothy R.
CORPORATE SOURCE: Department of Land Resources and Environmental Sciences, Montana State University, Bozeman, MT, 59717, USA
SOURCE: J. Bacteriol. (1999), 181(7), 2217-2224
CODEN: JOBAAY; ISSN: 0021-9193
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Recent work in this lab. has shown that the gene coding for acetate kinase (ackA) in Sinorhizobium meliloti is up-regulated in response to phosphate limitation. Characterization of the region surrounding ackA revealed that it is adjacent to pta, which codes for phosphotransacetylase, and that these two genes are part of an operon composed of at least two addnl. genes in the following order: an open reading frame (orfA), pta, ackA, and the partial sequence of a gene with an inferred peptide that has a high degree of homol. to **enoyl-ACP reductase** (fabI). Expts. combining enzyme assays, a chromosomal lacZ::ackA transcriptional fusion, complementation anal. with cosmid subclones, and the creation of mutations in pta and ackA all indicated that the orfA-pta-ackA-fabI genes are cotranscribed in response to phosphate starvation. Primer extension was used to map the position of the phosphate starvation-inducible transcriptional start sites upstream of orfA. The start sites were found to be preceded by a sequence having similarity to PHO boxes from other phosphate-regulated genes in S.

meliloti and to the consensus PHO box in **Escherichia coli**. Introduction of a phoB mutation in the wild-type strain eliminated elevated levels of acetate kinase and phosphotransacetylase activities in response to phosphate limitation and also eliminated the phosphate stress-induced upregulation of the ackA::lacZ fusion. Mutations in either ackA alone or both pta and ackA did not affect the nodulation or nitrogen fixation phenotype of S. meliloti.

REFERENCE COUNT: 58
REFERENCE(S):
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 11 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1999:118287 HCAPLUS
DOCUMENT NUMBER: 130:293235
TITLE: Molecular genetic analysis of enoyl-acyl carrier protein reductase inhibition by diazaborine
AUTHOR(S): De Boer, Gert-Jan; Pielage, Gerlof J. A.; Nijkamp, H. John J.; Slabas, Antoni R.; Rafferty, John B.; Baldock, Clair; Rice, David W.; Stuitje, Antoine R.
CORPORATE SOURCE: Department of Genetics, Institute for Molecular Biological Sciences, Vrije Universiteit, Amsterdam, 1081 HV, Neth.
SOURCE: Mol. Microbiol. (1999), 31(2), 443-450
CODEN: MOMIEE; ISSN: 0950-382X
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Diazaborine and isoniazid are, at first sight, unrelated anti-bacterial agents that inhibit the **enoyl-ACP reductase** (ENR) of **Escherichia coli** and Mycobacterium tuberculosis resp. The crystal structures of these enzymes including that of the diazaborine-inhibited E. **coli** ENR have been obtained at high resoln. Site-directed mutagenesis was used to study the importance of amino acid residues in diazaborine susceptibility and enzyme function. The results show that drug binding and inhibition require the presence of a glycine residue at position 93 of E. **coli** ENR or at the structurally equiv. position in the plant homolog, which is naturally resistant to the drug. The data confirm the hypothesis that any amino acid side-chain other than hydrogen at this position within the three-dimensional structure of these enzymes will affect diazaborine resistance by encroaching into the drug binding site. Substitutions of Gly-93 by amino acids with small side-chains, such as serine, alanine, cysteine and valine, hardly affected the catalytic parameters and rendered the bacterial host resistant to the drug. Larger amino acid side-chains, such as that of arginine, histidine, lysine and glutamine, completely inactivated the activity of the enzyme.

REFERENCE COUNT: 28
REFERENCE(S):
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 12 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:805899 HCAPLUS
DOCUMENT NUMBER: 130:193389
TITLE: Molecular structure of a reductase component of fatty

acid synthase
AUTHOR(S): Baldock, C.; Rafferty, J. B.; Stuitje, A. R.; Rice, D. W.
CORPORATE SOURCE: Krebs Institute for Biomolecular Research, Department of Molecular Biology and Biotechnology, The University of Sheffield, Sheffield, S10 2TN, UK
SOURCE: Soc. Exp. Biol. Semin. Ser. (1998), 67(Plant Lipid Biosynthesis), 73-92
CODEN: SEBSDI; ISSN: 0309-6831
PUBLISHER: Cambridge University Press
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review, with 30 refs., of the structure of the **enoyl acyl carrier protein reductase** component of fatty acid synthase from Brassica napus and **Escherichia coli**.
REFERENCE COUNT: 30
REFERENCE(S): (1) Baldock, C; Science 1996, V274, P2107 HCAPLUS
(2) Banerjee, A; Science 1994, V263, P227 HCAPLUS
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 13 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:588731 HCAPLUS
DOCUMENT NUMBER: 129:328141
TITLE: Mechanisms of isoniazid resistance in Mycobacterium tuberculosis: enzymic characterization of enoyl reductase mutants identified in isoniazid-resistant clinical isolates
AUTHOR(S): Basso, Luiz A.; Zheng, Renjian; Musser, James M.; Jacobs, William R., Jr.; Blanchard, John S.
CORPORATE SOURCE: Department of Biochemistry and Howard Hughes Medical Institute., Albert Einstein College of Medicine, Bronx, NY, 10461, USA
SOURCE: J. Infect. Dis. (1998), 178(3), 769-775
CODEN: JIDIAQ; ISSN: 0022-1899
PUBLISHER: University of Chicago Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mutants in the structural gene of the inhA-encoded NADH-dependent 2-trans **enoyl-acyl carrier protein reductase** were identified from isoniazid-resistant clin. isolates of Mycobacterium tuberculosis. Recombinant InhA proteins with defined single amino acid replacements were expressed in **Escherichia coli** and purified to homogeneity. Steady-stake kinetic parameters for wild type (WT) and I16T, I21V, I47T, and I95P mutants of the enoyl reductase were measured spectrophotometrically. NADH binding to WT and I16T, I21V, I47T, S94A, and I95P mutant reductases were detd. by fluorescence spectroscopy and demonstrated that all mutant enzymes had reduced NADH affinity and that NADH binding to all mutants was cooperative as compared with the hyperbolic binding of NADH to the WT enzyme. Since KatG-produced electrophilic derivs. of isoniazid have been suggested to inactivate the enoyl reductase-NADH complex, the kinetics of inactivation for the WT and 121V and I95P mutants was detd. Both mutations resulted in significantly increased values for the apparent first-order rate const. of inactivation.

L12 ANSWER 14 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:538134 HCAPLUS
DOCUMENT NUMBER: 129:228017
TITLE: Triclosan targets lipid synthesis

AUTHOR(S): McMurry, Laura M.; Oethinger, Margret; Levy, Stuart B.
CORPORATE SOURCE: Center for Adaptation Genetics and Drug Resistance, and Department of Molecular Biology and Microbiology, Tufts University School of Medicine, Boston, MA, 02111, USA
SOURCE: Nature (London) (1998), 394(6693), 531-532
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Macmillan Magazines
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Triclosan is a broad-spectrum antimicrobial. Here it is shown that triclosan blocks lipid synthesis in **Escherichia coli** and that mutations in, or overexpression of, gene fabI (which encodes **enoyl-acyl carrier protein reductase**, involved in fatty acid synthesis) prevents this blockage.

L12 ANSWER 15 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:360230 HCAPLUS
DOCUMENT NUMBER: 129:92174
TITLE: Crystallization of the NADP-dependent .beta.-keto acyl carrier protein reductase from Escherichia coli
AUTHOR(S): Rafferty, John B.; Fisher, Martin; Langridge, Sarah J.; Martindale, Wayne; Thomas, Neil C.; Simon, J. William; Bithell, Sian; Slabas, Antoni R.; Rice, David W.
CORPORATE SOURCE: Krebs Institute for Biomolecular Research, Department of Molecular Biology and Biotechnology, The University of Sheffield, Sheffield, S10 2TN, UK
SOURCE: Acta Crystallogr., Sect. D: Biol. Crystallogr. (1998), D54(3), 427-429
CODEN: ABCRE6; ISSN: 0907-4449
PUBLISHER: Munksgaard International Publishers Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB NADP-dependent .beta.-ketoacyl-[acyl carrier protein (ACP)] reductase (I) from E. **coli** was crystd. by the hanging-drop method of vapor diffusion using poly(ethylene glycol) of av. mol. wt. 1450. The I crystals belonged to hexagonal space group P6122 or P6522 with unit-cell dimensions a = b = 67.8, c = 355.8 .ANG.. Calcd. values for Vm and consideration of the packing suggested that the asym. unit contained a dimer. I catalyzes the 1st reductive step in the elongation cycle of fatty-acid biosynthesis. I shares extensive sequence homol. with the enzyme which catalyzes the 2nd reductive step in the cycle, **enoyl -[ACP] reductase**, and thus provides an opportunity to study the evolution of enzyme function in a metabolic pathway. The structure detn. will permit the anal. of the mol. basis of its catalytic mechanism and substrate specificity.

L12 ANSWER 16 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:289341 HCAPLUS
DOCUMENT NUMBER: 129:49033
TITLE: Mechanism of action of diazaborines
AUTHOR(S): Baldock, Clair; De Boer, Gert-Jan; Rafferty, John B.; Stuitje, Antoine R.; Rice, David W.
CORPORATE SOURCE: Krebs Institute for Biomolecular Research, Department of Molecular Biology and Biotechnology, The University of Sheffield, Sheffield, S10 2TN, UK
SOURCE: Biochem. Pharmacol. (1998), 55(10), 1541-1549
CODEN: BCPCA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 29 refs. The diazaborine family of compds. have antibacterial properties against a range of Gram-neg. bacteria. Initially, this was thought to be due to the prevention of

lipopolysaccharide synthesis. More recently, the mol. target of diazaborines has been identified as the NAD(P)H-dependent **enoyl acyl carrier protein reductase** (ENR), which catalyzes the last reductive step of fatty acid synthase. ENR from Mycobacterium tuberculosis is the target for the front-line antituberculosis drug isoniazid. The emergence of isoniazid resistance strains of M. tuberculosis, a chronic infectious disease that already kills more people than any other infection, is currently causing great concern over the prospects for its future treatment, and it has reawakened interest in the mechanism of diazaborine action. Diazaborines only inhibit ENR in the presence of the nucleotide cofactor, and this has been explained through the anal. of the x-ray crystallog. structures of a no. of **Escherichia coli** ENR-NAD+-diazaborine complexes that showed the formation of a covalent bond between the boron atom in the diazaborines and the 2'-hydroxyl of the nicotinamide ribose moiety that generates a noncovalently bound bisubstrate analog. The similarities in catalytic chem. and in the conformation of the nucleotide cofactor across the wider family of NAD(P)-dependent oxidoreductases suggest that there are generic opportunities to mimic the interactions seen here in the rational design of bisubstrate analog inhibitors for other NAD(P)H-dependent oxidoreductases.

L12 ANSWER 17 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:15665 HCAPLUS
DOCUMENT NUMBER: 128:99300
TITLE: Crystalline gene inhA enoyl-ACP reductase of Mycobacterium tuberculosis
INVENTOR(S): Sacchettini, James; Blanchard, John; Jacobs, Jr William R.
PATENT ASSIGNEE(S): Albert Einstein College of Medicine of Yeshiva University, USA
SOURCE: U.S., 22 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5702935	A	19971230	US 1994-234011	19940428
US 5648392	A	19970715	US 1995-386917	19950207
US 5556778	A	19960917	US 1995-491146	19950616
US 5837480	A	19981117	US 1996-700306	19960821
US 5882878	A	19990316	US 1996-701062	19960821
US 5837732	A	19981117	US 1996-766273	19961213
PRIORITY APPLN. INFO.:			US 1994-234011	19940428
			US 1994-307376	19940916
			US 1995-386917	19950207
			US 1995-491146	19950616
			US 1996-598085	19960207

AB Inha enzyme crystals and methods of growing said crystals are presented. Three crystal forms of the Inha enzyme with discrete unit cell parameters were obtained. The crystals of the Inha enzyme are of sufficient size and quality for x-ray crystallog. detn. of the three dimensional structure of the Inha enzyme in concert with heavy atom derivs. of said crystals. With the three dimensional structure of the Inha enzyme, compds. which inhibit the biochem. activity of the Inha enzyme may be developed. The M. tuberculosis **enoyl-ACP reductase** gene inhA was expressed in **Escherichia coli**. The recombinant enzyme was crystd. and its structure detd. by X-ray crystallog.

L12 ANSWER 18 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1997:594600 HCAPLUS
DOCUMENT NUMBER: 127:272787
TITLE: Antimicrobial activity of gemfibrozil and related

compounds
INVENTOR(S): Kabbash, Christina; Shuman, Howard A.; Silverstein, Samuel C.; Della-Latta, Phyllis
PATENT ASSIGNEE(S): Trustees of Columbia University in the City of New York, USA; Kabbash, Christina; Shuman, Howard A.; Silverstein, Samuel C.; Della-Latta, Phyllis
SOURCE: PCT Int. Appl., 107 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9731530	A1	19970904	WO 1997-US3158	19970228
W: AU, CA, JP, MX, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9721928	A1	19970916	AU 1997-21928	19970228
EP 888049	A1	19990107	EP 1997-914817	19970228
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1996-608712 19960229
WO 1997-US3158 19970228

AB A method is provided for inhibiting growth of a bacterium which consists essentially of contacting the bacterium with gemfibrozil or a deriv. thereof (Markush included). The compd. is present in a concn. effective to inhibit growth of the bacterium. The bacterial infections to be inhibited may be those assocd. with Legionella pneumophila, Mycobacterium tuberculosis, Bacillus subtilis, Bacillus megaterium, Pseudomonas oleovorans, Alcaligenes eutrophus, Rhodococcus sp., Citrobacter freundi, Group A Streptococcus sp., coag.-neg. Staphylococcus aureus, or Nocardia sp. Methods using the compds. of the invention for inhibiting enoyl reductase and for altering bacterial fatty acid synthesis are also disclosed.

L12 ANSWER 19 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1997:560724 HCAPLUS
DOCUMENT NUMBER: 127:246860
TITLE: Recombinant antibody fragments that detect enoyl acyl carrier protein reductase in Brassica napus
AUTHOR(S): Ziegler, Angelika; Macintosh, Sybil M.; Torrance, Lesley; Simon, William; Slabas, Antony R.
CORPORATE SOURCE: Scottish Crop Research Institute, Dundee, DD2 5DA, UK
SOURCE: Lipids (1997), 32(8), 805-809
CODEN: LPDSAP; ISSN: 0024-4201
PUBLISHER: AOCS Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Purified Brassica napus **enoyl acyl carrier protein reductase** (ENR) was used to select specific antibodies from a library of antibody fragments, single-chain Fv (scFv), displayed on filamentous phage. Anal. of the selected clones by BstNI fingerprinting and nucleotide sequencing showed that the scFv were derived from three different human VH germline genes. The binding specificities were confirmed by Western blots and ELISA. The scFv prepns. reacted with B. napus ENR, but not with .beta.-keto reductase, nor enoyl reductase from **Escherichia coli**. Anal. of fragments generated by CNBr treatment indicates that the scFv 3.13 recognizes an epitope located within the N-terminal 80 amino acids of the enzyme mol. The scFv were used to detect ENR directly in exts. of B. napus seeds.

L12 ANSWER 20 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1997:83769 HCAPLUS
DOCUMENT NUMBER: 126:128506
TITLE: Soluble and membrane bound components of plant lipid

synthesis
AUTHOR(S): Slabas, Antoni R.; Brown, Adrian P.; Rafferty, John B.; Rice, David W.; Baldock, Clare; Kroon, Johan T.M.; Simon, William; Stuitje, Antoine R.; Brough, Clare L.
CORPORATE SOURCE: Lipid Molecular Biology Group, Department of Biological Sciences, University of Durham, Durham, DH1 3LE, UK
SOURCE: C. R. Acad. Sci., Ser. III (1996), 319(11), 1043-1047
CODEN: CRASEV; ISSN: 0764-4469
PUBLISHER: Libbey Eurotext
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 13 refs. **Enoyl ACP reductase** (ENR) catalyzes the NADH dependent redn. of trans enoyl ACP to from satd. acyl ACPs; it is an essential component of the multisubunit type II fatty acid synthetase which is highly expressed in a temporal specific manner in seeds. The enzyme has been purified from rape, extensively sequenced, its cDNA cloned, and the protein overexpressed and crystd. The complete 3-dimensional structure of the enzyme has been detd. at 1.9 .ANG.. Difference Fourier anal. has shown that crotonyl ACP is a better substrate than crotonyl CoA as the latter also binds to the NADH pocket of the enzyme and thereby acts as an enzyme inhibitor. The potential active site has been identified from the position of conserved residues and by the location of the position of the nicotinamide ring of the NADH. In addn. extensive structural similarity has been found between ENR and the 3.alpha.-20.beta.-hydroxysteroid dehydrogenase. This has provided insights into the catalytic mechanisms which are being tested by site directed mutagenesis. In an attempt to gain insight into membrane bound enzymes of lipid biosynthesis we have employed a complementation cloning technique in E. **coli** to isolate the membrane bound 2-acyltransferase from Limnanthes douglasii. One of these shows distinct substrate specificity differences to the E. **coli** 2-AT. Introduction of the cDNA encoding this 2-AT into a high erucic acid rape line has allowed the synthesis of trierucin in the transgenic seed. Anal. of the transgenes and other acyltransferases is in progress.

L12 ANSWER 21 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1996:610216 HCAPLUS
DOCUMENT NUMBER: 125:269277
TITLE: Crystalline InhA enzyme-NADH complex
INVENTOR(S): Sacchettini, James
PATENT ASSIGNEE(S): Albert Einstein College of Medicine of Yeshiva University, A Division of Yes, USA
SOURCE: U.S., 8 pp. Cont. of U.S. Ser. No. 307,378,abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5556778	A	19960917	US 1995-491146	19950616
US 5702935	A	19971230	US 1994-234011	19940428
US 5882878	A	19990316	US 1996-701062	19960821
PRIORITY APPLN. INFO.:			US 1994-234011	19940428
			US 1994-307376	19940916
			US 1995-491146	19950616

AB The crystd. complex of NADH and InhA enzyme (**enoyl-acyl carrier protein reductase**) from Mycobacterium tuberculosis is presented. InhA enzyme was overexpressed in a com. available **Escherichia coli** system utilizing the nucleic acid sequence of InhA, purified, and single crystals of up to 0.6 mm3 in size were grown at 19.degree.. The crystals were hexagonal in shape and were of the space group P6222 with unit cell dimensions of a = b = 100.1 .ANG., c = 140.4 .ANG., .alpha. = .beta. = 90.degree., .gamma. =

120.degree.. Two substructures were identified in the single-domain protein. The first substructure is a core .alpha./.beta. structure composed of 6 parallel .beta. strands surrounded and interwoven by four .alpha.-helixes, harboring the N-terminal section of the macromol. The second substructure is a C-terminal region, composed mainly of two .alpha.-helixes interconnected by a short loop. The 3-dimensional structure of InhA enzyme can by utilized to design inhibitors to the InhA enzyme and subsequent treatment with those inhibitors of infection by M. tuberculosis (no data).

L12 ANSWER 22 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1996:412562 HCAPLUS
DOCUMENT NUMBER: 125:81320
TITLE: Molecular mechanisms of drug resistance in Mycobacterium tuberculosis
AUTHOR(S): Blanchard, John S.
CORPORATE SOURCE: Dep. Biochem., Albert Einstein Coll. Med., Bronx, NY, 10461, USA
SOURCE: Annu. Rev. Biochem. (1996), 65, 215-239
CODEN: ARBOAW; ISSN: 0066-4154
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 127 refs. In spite of forty years of effective chemotherapy for tuberculosis, the mol. mechanisms of antibacterial compds. in Mycobacterium tuberculosis have only recently been revealed. Broad spectrum antibacterials, including streptomycin, rifampicin, and fluoroquinolones, have been demonstrated to act on the same targets in M. tuberculosis as they do in **Escherichia coli**. Resistance to these agents results from single mutagenic events that lead to amino acid substitutions in their target proteins. The mechanisms of action of the unique antitubercular drugs, including isoniazid, ethambutol, and pyrazinamide, have also recently been defined. Resistance to isoniazid can be caused either by mutations in the katG-encoded catalase-peroxidase, the enzyme responsible for drug activation, or by the mol. target, the inhA-encoded long chain **enoyl-ACP reductase**. Ethambutol appears to block specifically the biosynthesis of the arabinogalactan component of the mycobacterial cell envelope, and pyrazinamide has no known target. With the resurgence of tuberculosis and the appearance of strains which are multiply resistant to the above compds., present tuberculosis chemotherapies are threatened. New approaches to the treatment of multidrug-resistant tuberculosis are needed.

L12 ANSWER 23 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1995:706895 HCAPLUS
DOCUMENT NUMBER: 123:103611
TITLE: A molecular study on the functional relationship between prokaryotic and plant enoyl-ACP reductases
AUTHOR(S): Stuitje, Antoine R.; Kater, Martin M.; Nijkamp, H. John J.
CORPORATE SOURCE: Institute Molecular Biological Sciences, Vrije Universiteit, Amsterdam, 1081 HV, Neth.
SOURCE: Plant Lipid Metab., [Pap. Int. Meet. Plant Lipids], 11th (1995), Meeting Date 1994, 87-9. Editor(s): Kader, Jean-Claude; Mazliak, Paul. Kluwer: Dordrecht, Neth.
CODEN: 61OZAO
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review, with 4 refs., on the **Escherichia coli** **envM** gene that encodes the bacterial counterpart of plant **enoyl-ACP reductase**, replacement of the bacterial **envM** gene by a plant counterpart, and the effect of a hybrid plant-bacterial fatty acid synthetase complex on bacterial growth and fatty acid compn. of the phospholipids.

L12 ANSWER 24 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1995:672981 HCAPLUS
DOCUMENT NUMBER: 123:79243
TITLE: Regulation of malonyl-CoA metabolism by acyl-acyl carrier protein and .beta.-ketoacyl-acyl carrier protein synthases in Escherichia coli
AUTHOR(S): Heath, Richard J.; Rock, Charles O.
CORPORATE SOURCE: Dep. Biochem., St. Jude Children's Res. Hosp., Memphis, TN, 38101, USA
SOURCE: J. Biol. Chem. (1995), 270(26), 15531-8
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The cessation of phospholipid biosynthesis by the inhibition of the sn-glycerol-3-phosphate acyltransferase using a plsB mutant led to an accumulation of long-chain acyl-acyl carrier proteins (acyl-ACP) and the concomitant inhibition of de novo fatty acid biosynthesis in **Escherichia coli**. Malonyl-CoA did not accumulate when phospholipid and fatty acid synthesis was blocked. However, the inactivation of .beta.-ketoacyl-ACP synthases I and II with the antibiotic cerulenin triggered a large increase in the accumulation of malonyl-CoA following the cessation of phospholipid synthesis, illustrating that the .beta.-ketoacyl-ACP synthases were responsible for the degrdn. of malonyl-CoA in the presence of long-chain acyl-ACP. The acyl-ACP requirement for malonyl-CoA degrdn. activity was confirmed by shifting **enoyl-ACP reductase** mutants (fabI(Ts)) to the non-permissive temp., leading to the abrupt cessation of fatty acid synthesis and the accumulation of malonyl-CoA in the absence of cerulenin. Anal. of the ACP pool compn. before and after the temp. shift showed that the fabI block did not result in the accumulation of long-chain acyl-ACP. These data indicate a feedback regulatory loop that functions to recycle malonyl-CoA to acetyl-CoA following the down-regulation of fatty acid and phospholipid formation and provides a physiol. rational for the acyl-ACP-dependent, malonyl-ACP decarboxylase reaction catalyzed by .beta.-ketoacyl-ACP synthases I and II.

L12 ANSWER 25 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1995:380323 HCAPLUS
DOCUMENT NUMBER: 124:47644
TITLE: Methods and compositions for detecting and treating mycobacterial infections using an inhA gene
INVENTOR(S): Jacobs, William R., Jr.; Collins, Desmond Michael; Banerjee, Asesh; De Lisle, Geoffrey William; Wilson, Theresa Mary
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9426312	A1	19941124	WO 1994-US5344	19940512
W: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KG, KP, KR, KZ, LK, LU, LV, MD, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2162868	AA	19941124	CA 1994-2162868	19940512
AU 9469121	A1	19941212	AU 1994-69121	19940512
AU 690121	B2	19980423		
EP 707496	A1	19960424	EP 1994-917378	19940512
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 09501823	T2	19970225	JP 1994-525723	19940512

PRIORITY APPLN. INFO.: NZ 1993-247620 19930513
US 1993-62409 19930514
WO 1994-US5344 19940512

AB The embodiments of the invention are based upon the identification and characterization of genes that det. mycobacterial resistance to the antibiotic isoniazid (INH) and its analogs. These genes, termed inhA, encode a polypeptide, InhA, that is the target of action of mycobacteria for isoniazid. The sequences of wild-type INH-sensitive as well as allelic or mutant INH-resistant inhA genes and their operons are provided. Also provided are isolated InhA polypeptides of both the INH-resistant and INH-sensitive types. Thus, the sequences of the inhA operons were detd. for sensitive strains of M. tuberculosis, M. smegmatis and M. bovis and resistant strains of M. smegmatis and M. bovis. The operons contain 2 open reading frames, the second of which (inhA) encodes the resistance determinant. A single mutation of Ala94 (in INH-resistant strains) from Ser94 (in INH-sensitive strains) mediates the INH-resistance phenotype in M. smegmatis and in M. bovis. The wild-type inhA gene also conferred INH and ethionamide resistance when transferred on a multicopy plasmid vector to M. smegmatis and M. bovis BCG. The InhA protein showed significant sequence conservation with the **Escherichia coli** enzyme **EnvM**, and cell-free assays indicate that it may be involved in mycolic acid biosynthesis.

L12 ANSWER 26 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1995:206469 HCAPLUS
DOCUMENT NUMBER: 122:2201
TITLE: Developmental specific expression and organelle targeting of the Escherichia coli fabD gene, encoding malonyl coenzyme A-acyl carrier protein transacylase in transgenic rape and tobacco seeds
AUTHOR(S): Verwoert, Ira I. G. S.; van der Linden, Karin H.; Nijkamp, H. John; Stuitje, Antoine R.
CORPORATE SOURCE: Institute Molecular Biological Sciences, Vrije Universiteit, Amsterdam, 1081 HV, Neth.
SOURCE: Plant Mol. Biol. (1994), 26(1), 189-202
CODEN: PMBIDB; ISSN: 0167-4412
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In both plants and bacteria, de novo fatty acid biosynthesis is catalyzed by a type II fatty acid synthetase (FAS) system which consists of a group of eight discrete enzyme components. The introduction of heterologous, i.e. bacterial, FAS genes in plants could provide an alternative way of modifying the plant lipid compn. In this study the **Escherichia coli** fabD gene, encoding malonyl CoA-ACP transacylase (MCAT), was used as a model gene to investigate the effects of over-producing a bacterial FAS component in the seeds of transgenic plants. Chimeric genes were designed, so as not to interfere with the household activities of fatty acid biosynthesis in the earlier stages of seed development, and introduced into tobacco and rapeseed using the Agrobacterium tumefaciens binary vector system. A napin promoter was used to express the E. **coli** MCAT in a seed-specific and developmentally specific manner. The rapeseed **enoyl-ACP reductase** transit peptide was used successfully, as confirmed by immunogold labeling studies, for plastid targeting of the bacterial protein. The activity of the bacterial enzyme reached its max. (up to 55 times the max. endogenous MCAT activity) at the end of seed development, and remained stable in mature transgenic seeds. Significant changes in fatty acid profiles of storage lipids and total seed lipid content of the transgenic plants were not found. These results are in support of the notion that MCAT does not catalyze a rate-limiting step in plant fatty acid biosynthesis.

L12 ANSWER 27 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1994:429155 HCAPLUS
DOCUMENT NUMBER: 121:29155
TITLE: The mechanism of inhibition of fatty acid synthase by the herbicide diflufenican

AUTHOR(S): Ashton, I. A.; Abulnaja, K. O.; Pallett, K. E.; Cole, D. J.; Harwood, J. L.
CORPORATE SOURCE: Dep. Biochem., Univ. Wales Coll. Cardiff, Cardiff, CF1 1ST, UK
SOURCE: Phytochemistry (1994), 35(3), 587-90
CODEN: PYTCAS; ISSN: 0031-9422
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The bleaching herbicide diflufenican (N-(2,4-difluorophenyl)-2-[3-(trifluoromethyl)phenoxy]-3-pyridine-carboxaminde) has been shown to inhibit plant fatty acid synthase. The mechanism of this inhibition was studied further by measuring the activities of the reductase components of the Type II fatty acid synthase complexes from **Escherichia coli** and avocado (Persea americana) mesocarp. Diflufenican had no effect on .beta.-ketoacyl-ACP reductase activity, but competitively inhibited both NADH- and NADPH-dependent **enoyl-ACP reductases**. This result suggest that chems. based on the diflufenican structure may be potential herbicides by virtue of their inhibition of fatty acid synthesis.

L12 ANSWER 28 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1994:158659 HCAPLUS
DOCUMENT NUMBER: 120:158659
TITLE: inhA, a gene encoding a target for isoniazid and ethionamide in Mycobacterium tuberculosis
AUTHOR(S): Banerjee, Asesh; Dubnau, Eugenie; Quemard, Annaik; Balasubramanian, V.; Um, Kyung Sun; Wilson, Theresa; Collins, Des; de Lisle, Geoffrey; Jacobs, William, R., Jr.
CORPORATE SOURCE: Howard Hughes med. Inst., Albert Einstein Coll. Med., Bronx, NY, 10461, USA
SOURCE: Science (Washington, D. C., 1883-) (1994), 263(5144), 227-30
CODEN: SCIEAS; ISSN: 0036-8075
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Isoniazid (isonicotinic acid hydrazide, INH) is one of the most widely used antituberculosis drugs, yet its precise target of action on Mycobacterium tuberculosis is unknown. A missense mutation within the mycobacterial inhA gene was shown to confer resistance to both INH and ethionamide (ETH) in M. smegmatis and in M. bovis. The wild-type inhA gene also conferred INH and ETH resistance when transferred on a multicopy plasmid vector to M. smegmatis and M. bovis BCG. The InhA protein shows a significant sequence conservation with the **Escherichia coli** enzyme **EnvM**, and cell-free assays indicate that it may be involved in mycolic acid biosynthesis. These results suggest that InhA is likely a primary target of action for INH and ETH.

L12 ANSWER 29 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1993:555839 HCAPLUS
DOCUMENT NUMBER: 119:155839
TITLE: The role of the **envM** genes of **Escherichia coli** and **Salmonella typhimurium** in cell membrane biosynthesis
AUTHOR(S): Turnowsky, Friederike; Bergler, Helmut; Ingolic, Elisabeth
CORPORATE SOURCE: Inst. Mikrobiol., Graz, A-8010, Austria
SOURCE: FEMS Symp. (1993), 65(Bacterial Growth and Lysis), 197-203
CODEN: FEMSDW; ISSN: 0163-9188
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Inhibition of the **envM** gene product by incubation of the conditional **envM** mutant E. **coli** JP1111 at the nonpermissive temp. or by diazaborine treatment leads to pleiotropic

effects on cell membrane compn. One of the most pronounced effects is the inhibition of phospholipid biosynthesis. The changes in the membrane structure lead to cell death without apparent lysis of the cells. The characteristic morphol. changes are the retraction of the cytoplasmic membrane which leaves empty zones mainly at the poles of the cells. Overprodn. of the wild type **EnvM** protein in the **envM** mutant at the nonpermissive temp. initially complements the ts phenotype but finally causes cell lysis.

L12 ANSWER 30 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1993:464411 HCAPLUS
DOCUMENT NUMBER: 119:64411
TITLE: Sequences of the **envM** gene and of two mutated alleles in **Escherichia coli**
AUTHOR(S): Bergler, Helmut; Hoegenauer, Gregor; Turnowsky, Friederike
CORPORATE SOURCE: Inst. Mikrobiol., Karl-Franzens-Univ., Graz, A-8010, Austria
SOURCE: J. Gen. Microbiol. (1992), 138(10), 2093-100
CODEN: JGMIAN; ISSN: 0022-1287
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The nucleotide sequence of E. **coli envM** gene was detd. It codes for a protein of 262 amino acids. The sequences of the E. **coli** and **Salmonella typhimurium EnvM** proteins are 98% identical. Gene **envM** is preceded in E. **coli** by a 43-nucleotide-long structural element, termed 'box C,' which occurs in several E. **coli** operons between structural genes. This sequence element is totally absent in S. **typhimurium** . Gene **envM** was mapped at coordinate position 1366.8 kb of the phys. map of Y. Kohara et al. (1987). As in S. **typhimurium**, a Gly for Ser exchange at position 241 of the **EnvM** protein results in a temp.-sensitive growth phenotype. Comparison of the **EnvM** amino acid sequence with sequences available in databases showed significant homol. with the family of short-chain alc. dehydrogenases.

L12 ANSWER 31 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1992:646310 HCAPLUS
DOCUMENT NUMBER: 117:246310
TITLE: cDNA cloning and expression of Brassica napus **enoyl-acyl carrier protein reductase** in **Escherichia coli**
AUTHOR(S): Kater, Martin M.; Koningstein, Gregory M.; Nijkamp, H. John J.; Stuitje, Antoine R.
CORPORATE SOURCE: Dep. Genet., Vrije Univ., Amsterdam, 1081 HV, Neth.
SOURCE: Plant Mol. Biol. (1991), 17(4), 895-909
CODEN: PMBIDB; ISSN: 0167-4412
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The onset of storage lipid biosynthesis during seed development in the oilseed crop Brassica napus (rape seed) coincides with a drastic qual. and quant. change in fatty acid compn. During this phase of storage lipid biosynthesis, the enzyme activities of the individual components of the fatty acid synthase system increase rapidly. A rapid and simple purifn. procedure in described for the plastid-localized NADH-dependent **enoyl-acyl carrier protein reductase** from developing B. napus seed, based on its affinity towards the acyl carrier protein (ACP). The purified protein was N-terminally sequenced and used to raise a potent antibody prepn. Immuno-screening of a seed-specific .lambda.gt11 cDNA expression library resulted in the isolated of **enoyl-ACP reductase** cDNA clones. DNA sequence anal. of an apparently full-length cDNA clone revealed that the **enoyl-ACP reductase** mRNA is translated into a precursor protein with a putative 73 amino acid leader sequence which is removed during the

translocation of the protein through the plastid membrane. Expression studies in **Escherichia coli** demonstrated that the full-length cDNA clone encodes the authntic B. napus NADH-dependent **enoyl-ACP reductase**. Characterization of the **enoyl-ACP reductase** genes by Southern blotting shows that the allo-tetraploid B. napus contains two pairs of related **enoyl-ACP reductase** genes derived from the two distinct genes found in both its ancestors, Brassica oleracea and B. campestris. Northern blot anal. of **enoyl-ACP reductase** mRNA steady-state levels during seed development suggests that the increase in enzyme activity during the phase of storage lipid accumulation is regulated at the level of gene expression.

L12 ANSWER 32 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1982:468345 HCAPLUS
DOCUMENT NUMBER: 97:68345
TITLE: The prokaryotic nature of the fatty acid synthetase of developing Carthamus tinctorius L. (safflower) seeds
AUTHOR(S): Shimakata, Takashi; Stumpf, Paul K.
CORPORATE SOURCE: Dep. Biochem. Biophys., Univ. California, Davis, CA, 95616, USA
SOURCE: Arch. Biochem. Biophys. (1982), 217(1), 144-54
CODEN: ABBIA4; ISSN: 0003-9861
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The component activities of the fatty acid synthetase (I) system of developing safflower seeds were sepd. and characterized and the mol. organization of the system was detd. The purified activities included acetyl-CoA-ACP (acyl-carrier protein)- and malonyl-CoA-ACP transacylases, .beta.-ketoacyl-ACP synthetase and **reductase**, .beta.-hydroxyacyl-**ACP** dehydrase, and **enoyl-ACP reductase**. .beta.-Ketoacyl-ACP reductase preferentially utilized NADPH (Km, 16 .mu.M) as the H donor; the Km for acetoacetyl-ACP was 9 .mu.M. .beta.-Hydroxyacyl-ACP dehydrase had a Km of 12 .mu.M for crotonyl-ACP. **Enoyl-ACP reductase** was present in 2 forms, I and II, which differed in purifn. and enzymic properties. The study indicated that the I system of safflower seeds is nonassocd. and similar in nature to the prokaryotic system of **Escherichia coli**.

L12 ANSWER 33 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1982:451628 HCAPLUS
DOCUMENT NUMBER: 97:51628
TITLE: Fatty acid synthetase of Spinacia oleracea leaves
AUTHOR(S): Shimakata, Takashi; Stumpf, Paul K.
CORPORATE SOURCE: Dep. Biochem. Biophys., Univ. California, Davis, CA, 95616, USA
SOURCE: Plant Physiol. (1982), 69(6), 1257-62
CODEN: PLPHAY; ISSN: 0032-0889
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The mol. organization of the fatty acid synthetase (I) system in spinach (S. oleracea) leaves was examd. by a procedure similar to that employed for the safflower system (Carathamus tinctorius). The crude ext. contained all the component activities (acetyl-CoA [acyl-carrier protein (ACP)]transacylase, malonyl-CoA-[ACP] transacylase, .beta.-ketoacyl-[ACP] synthetase, .beta.-ketoacyl-[ACP] **reductase**, .beta.-hydroxyacyl-[**ACP**] dehydrase, and **enoyl-**[**ACP**] **reductase** I involved in the synthesis of fatty acids; **enoyl-**[**ACP**] **reductase** II present in safflower seeds ext. was not detected spectrophotometrically. The component enzymes were clearly sepd. from one another by chromatog. procedures, including affinity chromatog. The properties of .beta.-ketoacyl-[ACP] **reductase**, .beta.-hydroxyacyl-[**ACP**] dehydrase, and **enoyl-**[ACP] reducase I from spinach were compared with the same enzymes in safflower seeds and **Escherichia**

coli. The I system of spinach leaves, as well as that of safflower seeds, is nonassociated and similar to the E. **coli** system.

L12 ANSWER 34 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1982:402625 HCAPLUS
DOCUMENT NUMBER: 97:2625
TITLE: The characteristics of some components of the fatty acid synthetase system in the plastids from the mesocarp of avocado (Persea americana) fruit
AUTHOR(S): Caughey, Isaac; Kekwick, Roy G. O.
CORPORATE SOURCE: Dep. Biochem., Univ. Birmingham, Birmingham, UK
SOURCE: Eur. J. Biochem. (1982), 123(3), 553-61
CODEN: EJBCAI; ISSN: 0014-2956
DOCUMENT TYPE: Journal
LANGUAGE: English
AB NADH- and NADPH-specific 3-oxoacyl-[**acyl-carrier-protein**] **reductases**, **enoyl-**[**acyl-carrier-protein**] **reductase** (EC 1.3.1.9), and [acyl-carrier-protein] malonyltransferase (EC 2.3.1.39) were purified from prepns. of avocado mesocarp plastids and characterized. The enzymes were quite similar in mol. and kinetic characteristics to analogous enzymes known in **Escherichia coli** and Euglena and were clearly components of a type-II fatty acid synthetase system.

L12 ANSWER 35 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1982:118114 HCAPLUS
DOCUMENT NUMBER: 96:118114
TITLE: Stereochemical studies of fatty acid biosynthesis: steric positions of hydrogens incorporated into fatty acids
AUTHOR(S): Saito, Kazutoshi; Kawaguchi, Akihiko; Seyama, Yosuke; Yamakawa, Tamio; Okuda, Shigenobu
CORPORATE SOURCE: Inst. Appl. Microbiol., Univ. Tokyo, Tokyo, Japan
SOURCE: Tennen Yuki Kagobutsu Toronkai Koen Yoshishu, 24th (1981), 529-36. Osaka Univ., Fac. Pharm. Sci.: Suita, Japan.
CODEN: 47BNAB
DOCUMENT TYPE: Conference
LANGUAGE: Japanese
AB The steric course of the enoyl redn. catalyzed by fatty acid synthetases was investigated with 2H as a tracer. 2H-labeled decanoic acids were synthesized by **Escherichia coli enoyl-acyl carrier protein** (ACP) **reductase** from various 2H sources. Subsequent addn. of acyl-CoA oxidase catalyzed the anti elimination of the pro-2R and pro-3R H atoms of acyl-CoA. The 2H contents of satd. and 2,3-dehydro fatty acids were analyzed by gas chromatog.-mass spectroscopy. 2H-labeled fatty acids were synthesized from [2-2H2]malonyl-CoA or in the presence of stereospecifically 2H-labeled NAD(P)H by fatty acid synthetases from yeast, Brevibacterium ammoniagenes, and rat liver. Anal.of the steric positions of 2H atoms by the above method indicated that the H of NAD(P)H was incorporated via a 3-Si attack by E. **coli** and B. ammoniagenes fatty acid synthetases, and via a 3-Re attack by the rat liver enzyme. The H of H2O was incorporated via a 2-Re attack by E. **coli** and B. ammoniagenes enzymes, and via a 2-Si attack by yeast and rat liver enzymes. The stereochem. of the formation of oleic acid by B. ammoniagenes fatty acid synthetase was also investigated.

L12 ANSWER 36 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1980:421542 HCAPLUS
DOCUMENT NUMBER: 93:21542
TITLE: Incorporation of hydrogen atoms from deuterated water and stereospecifically deuterium-labeled nicotinamide nucleotides into fatty acids with the Escherichia coli fatty acid synthetase system

AUTHOR(S): Saito, Kazuki; Kawaguchi, Akihiko; Okuda, Shigenobu; Seyama, Yousuke; Yamakawa, Tamio
CORPORATE SOURCE: Fac. Med., Univ. Tokyo, Tokyo, Japan
SOURCE: Biochim. Biophys. Acta (1980), 618(2), 202-13
CODEN: BBACAQ; ISSN: 0006-3002
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The mechanism of H incorporation into fatty acids was investigated with intact E. **coli** cells, a crude enzyme prepn., and purified reductases of the fatty acid synthetase system. The distributions of 2H atoms incorporated into fatty acids from 2H2O and stereospecifically 2H-labeled NADPH or NADH were detd. by mass spectrometry. When E. **coli** was grown in 2H2O, almost every H atom of cellular fatty acids was incorporated from the medium. When fatty acids were synthesized from acetyl-CoA, malonyl-CoA, and NADPH in the presence of a crude enzyme prepn. of either E. **coli** or Bacillus subtilis, almost every H atom was also incorporated from the medium. In contrast to these results, purified .beta.-ketoacyl-acyl carrier protein reductase directly transferred the HB (H at the B side of the dihydropyridine ring) of NADPH to .beta.-ketoacyl-acyl carrier protein, and purified **enoyl acyl-carrier protein reductase** also transferred the HB of NADPH and NADH directly to enoyl acyl-carrier protein. In the crude enzyme prepn. of E. **coli**, high activities were found which exchanged the HB of NADPH with the 2H of 2H2O. The conflicting results of the origin of H atoms of fatty acids mentioned above are explained by the presence of enzymes which catalyze the rapid exchange of NADPH with the 2H of 2H2O prior to the reaction of fatty acid synthetase.

L12 ANSWER 37 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1970:410827 HCAPLUS
DOCUMENT NUMBER: 73:10827
TITLE: **Enoyl acyl carrier protein reductases** from **Escherichia coli**
AUTHOR(S): Weeks, Gerald; Wakil, Salih J.
CORPORATE SOURCE: Med. Center, Duke Univ., Durham, N. C., USA
SOURCE: Methods Enzymol. (1969), 14, 66-73
CODEN: MENZAU
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The 2 step of fatty acid synthesis is the redn. of trans-.alpha.,.beta.-unsaturated acyl carrier protein (ACP) by DPNH or TPNH and is catalyzed by **enoyl-ACP reductase**. Crotonyl-ACP was used as the substrate and was synthesized from crotonic anhydride and ACP at pH 8 in the presence of an equiv. amt. of dithiothreitol. The enzyme was obtained from E. **coli** in 20% yield and 250-fold purification. The activity ratio for TPNH:DPNH varied between 1.6 and 2.8 during purification. This TPNH and DPNH reductase mixt. was stable at 55.degree. for 5 min but not at 60.degree.. The TPNH-dependent reaction had an optimum range of pH 6-9. The DPNH-dependent reductase was active with ACP and CoA derivs. (Km = 40.mu.M for crotonyl-ACP and 2.5 mM for crotonyl-CoA) while the TPNH-dependent reductase was specific for ACP derivs. The TPNH-dependent activity had a higher activity with short chain acyl-ACP derivs.; both enzymes showed max. activities with 2-hexenoyl derivs. Thiol alkylating agents inhibited both enzymes. N-ethylmaleimide at 1 mM inhibited the TPNH enzyme 66% and stimulated the DPNH activity 2.5 times.

L12 ANSWER 38 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1970:28471 HCAPLUS
DOCUMENT NUMBER: 72:28471
TITLE: Mechanism of fatty acid synthesis. XXII. Salt activation of the fatty acid-synthesizing enzymes of Escherichia coli
AUTHOR(S): Schulz, Horst; Weeks, Gerald; Toomey, Richard E.;

Shapiro, Martin; Wakil, Salih J.
CORPORATE SOURCE: Med. Center, Duke Univ., Durham, N. C., USA
SOURCE: J. Biol. Chem. (1969), 244(24), 6577-83
CODEN: JBCHA3
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Salts activate the fatty acid-synthesizing system of E. **coli** and severa l of its component enzymes. The 3-hydroxyacyl CoA dehydrogenase from pig heart, one of the enzymes o f fatty acid oxidn., is also stimulated by salts. Although all cations examd. produce this stimulation, the activation patte rns caused by monovalent cations are clearly distinct from those of diva lent cations. When acyl carrier protein (ACP) and CoA thioesters are used as substrates, the activities of the .beta.-ketoacyl-ACP **reductase** and the **enoyl-ACP reductase** are stimulated by salts. However, there is no stimulation when N-acetylcysteamine thioesters serve as substrates. With the .beta.-ketoacyl-ACP reductase an increase of the salt concn. causes an increase in Vmax. for both acetoacetyl-ACP and acetoacetyl-CoA and a decrease in Km only for acetoacetyl-ACP. The chromatographic behavior of ACP on Sephadex G-100 is influenced by salts. ACP interacts with MgSO4 to form complexes which elute in earlier fractions from Sephadex G-100 than salt-free ACP, although the mol. wt. of the ACP is unchanged. When acetoacetyl-ACP, complexed with MgSO4, is used as substrate for the .beta.-ketoacyl-ACP reductase, an increase of the salt concn. increases the Vmax. for the reaction but has no effect on the Km for the acetoacetyl-ACP-MgSO4 complex. It is suggested that cations complex with the protein moiety of ACP substrates, thereby facilitating binding to the enzymes, and that cations increase reaction rates possibly by reducing the repulsion between the neg. charged groups of the substrates and their resp. enzymes.

L12 ANSWER 39 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1968:424753 HCAPLUS
DOCUMENT NUMBER: 69:24753
TITLE: Studies on the mechanism of fatty acid synthesis. XX. Preparation and general properties of .beta.-hydoxybutyryl acyl carrier protein dehydrase
AUTHOR(S): Mizugaki, Michinao; Weeks, Gerald; Toomey, Richard E.; Wakil, Salih J.
CORPORATE SOURCE: Med. Center, Duke Univ., Durham, N. C., USA
SOURCE: J. Biol. Chem. (1968), 243(13), 3661-70
CODEN: JBCHA3
DOCUMENT TYPE: Journal
LANGUAGE: English
AB An enzyme, .beta.-hydroxybutyryl acyl carrier protein (ACP) dehydrase, which catalyzes the reversible dehydration of short chain .beta.-hydroxyacyl-ACP to the corresponding .alpha.,.beta.-unsatd. acyl-ACP derivs. was isolated from exts. of **Escherichia coli**. Dehydrase prepns. of specific activity of 5000 nanomoles/min./mg. of protein were homogeneous. The enzyme has an estd. mol. wt. of 26,000. It is relatively heat-stable and is active over a broad pH range with max. activity between pH 7.5 and 8.5. The dehydrase has a functional SH group and can be readily inhibited by SH-binding reagents such as N-ethylmaleimide and iodoacetamide. The dehydrase reaction is readily reversible and the equil. const. for the dehydration of .beta.-hydroxybutyryl-ACP is estd. to be 19M. The .beta.-hydroxybutyryl-ACP dehydrase is active on ACP thioesters but inactive on the CoA derivs. The enzyme is specific for short chain length .beta.-hydroxyacyl-ACP derivs. (C4 to C3). The estd. max. rates of hydration of crotonyl-ACP, 2-hexenoyl-ACP, and 2-octenoyl-ACP are 4100, 2300, and 200 nanomoles/min./mg., resp. The enzyme is inactive on 2-decenoyl-ACP. This limited chain length specificity of the .beta.-hydroxybutyryl-ACP dehydrase is responsible for the accumulation of .beta.-hydroxydecanoyl-ACP in a reconstituted fatty acid-synthesizing system comprised of highly purified prepns. of malonyl-CoA-ACP transacylase, acyl-malonyl-ACP-condensing enzyme, .beta.-ketoacyl-ACP

reductase, .beta.-hydroxybutyryl-**ACP** dehydrase, and **enoyl-ACP reductase**. The .beta.-hydroxydecanoyl-ACP thus synthesized is readily converted to either satd. or unsatd. fatty acids by the action of protein fractions which contain at least 2 long-chain .beta.-hydroxyacyl-ACP dehydrases.

L12 ANSWER 40 OF 40 HCAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1968:75320 HCAPLUS
DOCUMENT NUMBER: 68:75320
TITLE: Mechanism of fatty acid synthesis. XVIII. Preparation and general properties of the **enoyl acyl carrier protein reductases** from **Escherichia coli**
AUTHOR(S): Weeks, Gerald; Wakil, Salih J.
CORPORATE SOURCE: Duke Univ. Med. Center, Durham, N. C., USA
SOURCE: J. Biol. Chem. (1968), 243(6), 1180-9
CODEN: JBCHA3
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Prepns. of **enoyl acyl carrier protein** (ACP) **reductase** have been purified 250-fold over crude exts. of E. **coli**. Such prepns. catalyze the redn. of .alpha.,.beta.-unsatd.-acyl ACP to satd. derivs. They utilize both TPNH and DPNH as electron donors for the redn. of the substrate. Available evidence indicates that the TPNH-dependent and the DPNH-dependent reductase activities are due to 2 distinct enenzymes, a TPNH **enoyl -ACP reductase** and a DPNH **enoyl-ACP reductase**. The TPNH enoyl reductase is unstable at higher pH values (>7.50) and is enzymically inactive above pH 8.0. It exhibits an abs. specificity for the acyl ACP substrates and is more active on crotonyl ACP than on longer-chain .alpha.,.beta.-unsaturated-acyl ACP. It has a functional thiol group and can be readily inhibited by p-hydroxymercuribenzoate, iodoacetate, and N-ethylmaleimide. In contrast, the DPNH **enoyl-ACP reductase** is relatively stable at higher pH values and is enzymically active over a wide range of pH values. It utilizes both acyl ACP and acyl CoA substrates and exhibits higher activity with decenoyl ACP than with crotonyl ACP. It has a functional thiol group and can be readily inhibited by p-hydroxymercuribenzoate and iodoacetate, but not by N-ethylmaleimide, which causes 2-to 3-fold stimulation of enzymic activity. The reductase reaction is essentially irreversible, and the stoichiometry of the reaction is consistent with the formulation that 1 mole of reduced pyridine nucleotide is consumed for every mole of .alpha.,.beta.-unsatd.-acyl ACP reduced. Evidence is presented that the fatty acid-synthesizing system of E. **coli** requires both TPNH and DPNH for max. activity. The requirement for DPNH is at the reductase step, and both nucleotides have a synergistic effect in total fatty acid synthesis. 19 references.

=> SELECT HIT RN L6 1-21

E1 THROUGH E11 ASSIGNED

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=> S E1-E11

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             1 37251-08-4/BI
               (37251-08-4/RN)
             1 148998-18-9/BI
               (148998-18-9/RN)
             1 126730-36-7/BI
               (126730-36-7/RN)
             1 126730-38-9/BI
               (126730-38-9/RN)
             1 126731-07-5/BI
               (126731-07-5/RN)
             1 126731-08-6/BI
               (126731-08-6/RN)
             1 148998-19-0/BI
               (148998-19-0/RN)
             1 148998-20-3/BI
               (148998-20-3/RN)
             1 187857-08-5/BI
               (187857-08-5/RN)
             1 200385-48-4/BI
               (200385-48-4/RN)
             1 239448-02-3/BI
               (239448-02-3/RN)
L13         11 (37251-08-4/BI OR 148998-18-9/BI OR 126730-36-7/BI OR 126730-38-
               9/BI OR 126731-07-5/BI OR 126731-08-6/BI OR 148998-19-0/BI OR
               148998-20-3/BI OR 187857-08-5/BI OR 200385-48-4/BI OR 239448-02-
               3/BI)
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=>

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=> D IDE CAN L13 1-11

L13 ANSWER 1 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **239448-02-3** REGISTRY
CN Dehydrogenase, short-chain alcohol (Mannheimia haemolytica strain OSU gene envM) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN GenBank AF033119-derived protein GI 2645727
CN Short-chain alcohol dehydrogenase (Pasteurella haemolytica strain OSU gene envM)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 131:180620

L13 ANSWER 2 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **200385-48-4** REGISTRY
CN DNA (Mannheimia haemolytica strain OSU gene fnrP plus gene envM plus flanks) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN DNA (Pasteurella haemolytica strain OSU gene fnrP plus gene envM plus flanks)
CN GenBank AF033119
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR GenBank
LC STN Files: CA, CAPLUS, GENBANK, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 131:180620

L13 ANSWER 3 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **187857-08-5** REGISTRY
CN Reductase, enoyl- [acyl carrier protein] (Escherichia coli strain K-12 clone Kohara-#255 gene fabI) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN GenBank D90766-derived protein GI 1742101
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 126:207976

L13 ANSWER 4 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **148998-20-3** REGISTRY
CN Protein (Escherichia coli clone pFT617 gene envM reduced) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN NADH-dependent enoyl-acyl carrier protein reductase variant (Escherichia coli JP1111 temp.-sensitive mutant gene fabI)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 120:318216

L13 ANSWER 5 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **148998-19-0** REGISTRY
CN Protein (Escherichia coli clone pFT650 gene envM reduced) (9CI) (CA INDEX NAME)
OTHER NAMES:

CN NADH-dependent enoyl-acyl carrier protein reductase variant (Escherichia coli gene fabI diazaborine resistance-conferring)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 120:318216

L13 ANSWER 6 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **148998-18-9** REGISTRY
CN Protein (Escherichia coli clone pHAP1 gene envM reduced) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN enoyl-ACP reductase (Escherichia coli clone pEAR3 gene envM)
CN GenBank AE000227-derived protein GI 1787545
CN NADH-dependent enoyl-acyl carrier protein reductase (Escherichia coli gene fabI) (E.C. 1.3.1.9)
CN Reductase, enoy. [acyl carrier protein] (Escherichia coli clone pEAR3 gene envM)
CN Reductase, enoyl- [acyl carrier protein] (Escherichia coli strain K12-MG1655 gene fabI)
CN Reductase, enoyl- [acyl carrier protein] (Escherichia coli gene fabI)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
5 REFERENCES IN FILE CA (1967 TO DATE)
5 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 131:134684

REFERENCE 2: 128:44390

REFERENCE 3: 122:73533

REFERENCE 4: 120:318216

REFERENCE 5: 119:64411

L13 ANSWER 7 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **126731-08-6** REGISTRY
CN Protein (Salmonella typhimurium clone pKF403 gene envM reduced) (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 112:192764

L13 ANSWER 8 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **126731-07-5** REGISTRY
CN Protein (Salmonella typhimurium clone pFT501 gene envM reduced) (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 112:192764

L13 ANSWER 9 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **126730-38-9** REGISTRY
CN DNA (Salmonella typhimurium clone pKF403 gene envM) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Deoxyribonucleic acid (Salmonella typhimurium clone pKF403 gene envM)
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 112:192764

L13 ANSWER 10 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **126730-36-7** REGISTRY
CN DNA (Salmonella typhimurium clone pFT501 gene envM) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Deoxyribonucleic acid (Salmonella typhimurium clone pFT501 gene envM)
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 112:192764

L13 ANSWER 11 OF 11 REGISTRY COPYRIGHT 2000 ACS
RN **37251-08-4** REGISTRY
CN Reductase, enoyl- [acyl carrier protein] (9CI) (CA INDEX NAME)
OTHER NAMES:
CN E.C. 1.3.1.9
CN Enoyl-ACP reductase
CN Enoyl-[acyl carrier protein] reductase
CN NADH-dependent enoyl acyl carrier protein reductase
CN NADH-enoyl acyl carrier protein reductase
CN NADH-enoyl-ACP reductase
CN NADH-specific enoyl-ACP reductase
MF Unspecified

CI MAN
LC STN Files: AGRICOLA, ANABSTR, BIOSIS, CA, CAPLUS, CEN, TOXLIT, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
117 REFERENCES IN FILE CA (1967 TO DATE)
6 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
118 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 133:306308

REFERENCE 2: 133:291952

REFERENCE 3: 133:220072

REFERENCE 4: 133:161156

REFERENCE 5: 133:147581

REFERENCE 6: 133:146752

REFERENCE 7: 132:344846

REFERENCE 8: 132:260229

REFERENCE 9: 132:204092

REFERENCE 10: 132:146212